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An Address.[1]

THE ASSOCIATION, THE COLLEGES, AND THE FUTURE

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President, New South Wales Branch of the British Medical Association

As the subject of my address tonight I have chosen the problem of our Association, its relationship to the Colleges, and the future. It may be temerous on my part to open this subject, and yet it is one to be faced, and little has been addressed to our members concerning it.

If, seeking guidance, we look at the course of events in Britain, it is significant that as early as 1518 the College of Physicians was active in maintaining and advancing medical education, controlling ethical conduct within the profession and acting as an advisory body to the Crown and Parliament. Subsequently, the College of Surgeons was founded, and at a much later date the British Medical Association; then came the College of Obstetricians and Gynæcologists, and only some six years ago the College of General Practitioners. It is not unreasonable that a body established over 400 years ago should feel responsible for the welfare of its Fellows and Members when faced with medico-political or any other problems, and it is understandable that the younger Colleges would be influenced by the senior College in their approach to these matters.

It is important that the infant College of General Practitioners, now in its sixth year of life, has already a membership of some five thousand, and the Australian Faculty of some seven hundred members has seen fit to found an autonomous Australian College of General Practitioners, thus indicating that our general practitioner members do desire some additional service or activity which our Association does not supply or has not supplied adequately.

It is not remarkable that physicians and surgeons in Britain might turn to their College, to which they traditionally must belong, for guidance rather than to our parent body, but the position in Australia is different in that our Association is the oldest established body here and the one to which all but a small minority belong. Furthermore, it has served the profession faithfully and well, and alone has guided us through many troublous times. Until recent years it has provided all our needs, including our scientific wants, and by virtue of experience is still best fitted to guide us in many of our activities.

However, we must now recognize that the two Royal Australasian Colleges, the Chapter of the Royal College of

[1]Read at the Annual Meeting of the New South Wales Branch of the British Medical Association on March 27, 1958.

Obstetricians and Gynæcologists and more recently the Australian College of General Practitioners, as well as numerous other bodies, are playing an increasing part in post-graduate medical education. These organizations have taken over many of the former scientific activities of the Association, in addition to dealing with what we may term the domestic affairs of their members. It is inevitable that the physician or surgeon finds it increasingly difficult to maintain his interest in the Section of Medicine or the Section of Surgery when his own college is so active in his own particular sphere. Perhaps more importantly the general practitioner has found his young college vitally interested in post-graduate education and in stimulating clinical research within his own sphere.

As these various bodies grow in stature, how should the Association plan for the future? Should we attempt to integrate their protean activities? Above all how can we avoid split loyalties? These are some of the problems we face.

Scientifically for the present, I feel, as an Association we should continue to hold joint sectional meetings, where specialists' views on a common subject may be integrated for the benefit of all. In the past these meetings have been invaluable. Those sections which have not as yet formed special bodies must be supported, but particularly should we aim at maintaining the high standard of the scientific sessions of our Congresses, aiming at joint sessional activities. Clinical meetings in our hospitals, more especially in country hospitals, should be encouraged, and where required a specialist or specialists supplied to aid discussion. Adequate support must be given to the scientific activities of the Post-Graduate Committee and such organizations as F.O.C.L.A. Above all, we must maintain our Journal and our library service, which has reached such a high standard. If in the future the College of General Practitioners, the Post-Graduate Committee or any other body should supply an equally adequate or better service in any of these fields, we should be prepared to withdraw and to direct our energies into those many other spheres in which our Association is best fitted to lead and direct the profession, and to which I shall now make some reference.

Public relations have assumed an increasing importance in our professional life as we progress towards the Welfare State. The N.S.W. Branch has put much effort into this activity under the able chairmanship of Dr. Edgar Thomson. Unless the public is to be confused, there must be only one body responsible for this very important function; and as we are representative of all branches of the profession, it is obviously our task and our responsibility. It is doubtful if our members are yet fully aware of the significance of this activity or of its far-reaching effects in our fight for survival as a free profession.

The Association has always been deeply interested in medical education. As the one body which includes in its membership doctors interested in all branches of medicine, it should be possible for us to establish an advisory committee capable of coordinating the views of the academic teacher, the clinician and the administrator in planning the future. Preferably this should be at a Federal level, but for the moment let us see that the present State committee does function adequately and receives the support of members. It will not act in haste, and its function is constructive not destructive.

The ethical conduct of our profession must continue to remain our problem, and it is an ever-increasing one. Not only should the high standard of conduct between doctor and patient and doctor and doctor be maintained, but with the changing pattern of medical practice the ethical conduct of our members with respect to the Government, the insurance companies and similar bodies is assuming increasing importance. Some, admittedly a small minority, have not been seized with the necessity for impeccable honesty in dealing with these third parties. A failure to realize this fact could do the profession irreparable harm.

And now we come to what is perhaps one of the most important present-day and future responsibilities of this Association, namely, our medico-political activity. In this changing political economy of Australia we have fought for and have been permitted to retain at present our independence owing to the determined attitude of the Association and the constant vigilance of Federal and State Councils. In this field we remember with gratitude the outstanding services of some of our most distinguished members and in particular the late Sir Archibald Collins.

There will always be problems between Governments, insurance companies, hospital boards and similar bodies and ourselves, and it is of paramount importance that there be only one negotiating body, which should, I feel, be our Association through its Federal or State Councils. In this way, and only in this way, can we avoid split loyalties and those troubles which beset the profession in Britain today. It is equally important that the Association does truly represent the views of all branches of the profession and not one sectional interest. How best to safeguard this principle is not yet clear to me, but obviously there must be the closest cooperation between the Association and the Colleges. Divergent views, when they exist, must be settled amicably within the profession before negotiations with outside organizations reveal them and lead to further divergence.

In conclusion, it can be justly said that the foundation of the Royal Australasian Colleges and kindred bodies has represented a great advance in medicine in Australia. Without them our scientific standards could not have been maintained. The Association has achieved much in this field in the past, but with the changing way of professional life we should, with some reluctance perhaps, hand over many of these activities and direct our energies into other channels, to some of which I have just referred. Admittedly, some of these activities may be slightly distasteful to us as doctors and individuals concerned primarily with the welfare of our patients and the advancement of knowledge. Nevertheless, if we are to continue to enjoy that freedom which was the privilege of our fathers, and if we are to maintain our present status within the community, such activities are essential and very much the responsibility of our Association.

PREGNANCY IN THE DIABETIC.

By G. M. PARKIN, M.B., B.S., D.G.O., M.R.C.O.G.,
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BEFORE the discovery and introduction of insulin in 1922, a woman who developed diabetes had a comparatively short life expectancy, and rarely succeeded in becoming pregnant and producing a child. However, in these days when the outlook for the diabetic is so completely changed, it is quite commonplace for a diabetic woman to embark upon a pregnancy. The combination of pregnancy and diabetes presents a number of interesting clinical problems —namely: How is diabetes affected by pregnancy? What are the risks that severe diabetic complications will occur during pregnancy, labour and the puerperium? Does pregnancy permanently aggravate diabetes? How does diabetes affect pregnancy, labour and the puerperium? What obstetric complications occur in diabetes and how do they affect the maternal outcome? How can the large perinatal mortality rate be reduced?

The perinatal mortality rate is variously reported as from about 20% to 40%, very few series giving rates below 20%. The loss of at least one in five of all babies born to diabetic mothers presents one of the biggest problems in the management of pregnancy in diabetes, and I propose to confine my remarks mainly to the factors which contribute to this very high perinatal mortality.

Material.

The present series comprises all diabetic pregnancies with viable fœtuses at King George V Memorial Hospital, Sydney, from its opening in 1943 until the end of 1956.

In all there are 110 pregnancies in 76 patients, with a maternal mortality of two, or 1·8%, and a perinatal mortality of 23, or 20·9%. The series comprises a variety of patients—private patients attended by their own physician and obstetrician, public patients attending the general ante-natal clinic (prior to 1951), public patients attending the combined diabetic ante-natal clinic (after 1951), and non-booked patients admitted in emergency (Table I).

In general, the series has been investigated as a whole rather than according to the groups outlined above. Only

TABLE I.

Subdivision of the Series into Four Groups, with the Associated Fœtal Mortalities.

Patients.	Number of Cases.	Maternal Deaths.	Fœtal Deaths.	Fœtal Mortality.
Private patients	34	2	4	11·8%
Public patients:				
Before 1951	32	—	10	31·3%
After 1951	39	—	7	18·0%
Non-booked patients ..	5	—	2	40·0%

pregnancies proceeding beyond the twenty-eighth week have been included, as this has been the practice in most published series. The incidence of abortion amongst diabetic patients was not determined, but most authors (for example, Barns and Morgans, 1948; Clayton, 1956)

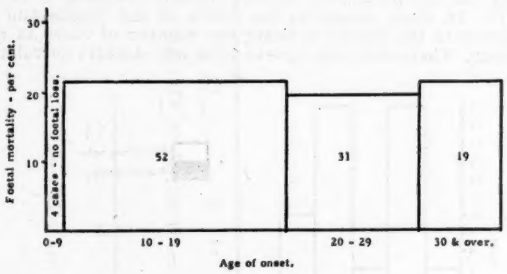

FIGURE I.

state that the abortion rate amongst diabetics is no higher than that amongst non-diabetic patients. Amongst the patients attending the combined diabetic ante-natal clinic the abortion rate was 12%, well within normal limits.

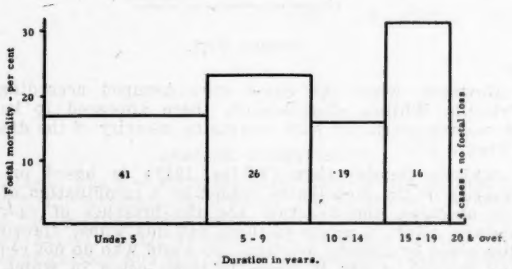

FIGURE II.

Maternal Complications.

Before I proceed to the consideration of fœtal mortality, maternal complications will be briefly considered. The main complications that occur in diabetic pregnancies are, first, purely obstetric ones—namely, toxæmia, hydramnios, and difficult labour due to a large fœtus—and, second, complications which can occur quite apart from pregnancy —namely, vascular disease in its various forms, diabetic acidosis, insulin reactions, etc. The important complications from the obstetric viewpoint are preeclampsia, hydramnios, hypertension and nephropathy. Difficult labour is now rarely encountered, because of modern views on the method of delivery.

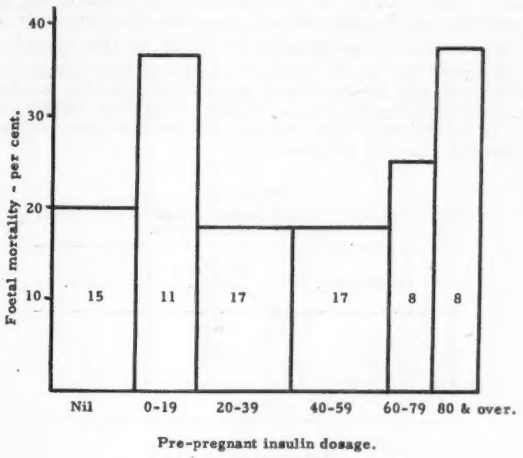

FIGURE III.

There is often difficulty in distinguishing between minor degrees of preeclampsia and diabetic nephropathy. Cases in which albuminuria occurs after the twentieth week, in the absence of any evidence of chronic nephritis or diabetic nephropathy, have been considered as preeclampsia.

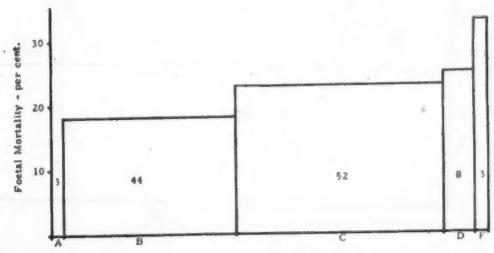

FIGURE IV.

The incidence of preeclampsia in this series was 47·7%. Widely different rates are reported, varying from 10% to 70%, and this wide variation is no doubt due to differences in the standards of diagnosis. However, it is generally

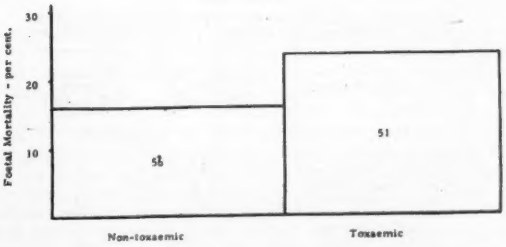

FIGURE V.

agreed that the incidence of toxæmia in diabetic patients is significantly higher than in non-diabetic patients. Hagbard (1956), from Sweden, found that toxæmia occurs more commonly as the pregnancy approaches term, and quite a large proportion of cases of toxæmia in his early

series occurred after the thirty-sixth week. Toxæmia is more common when the diabetes has been of long standing, particularly when it commenced at an early age.

Nephropathy was manifest in nine cases, or 8·2%. Once again, nephropathy is more common in diabetes of long standing, particularly when it commenced at an early age.

Hypertension was present in 12·7% of the cases.

Hydramnios as a clinical manifestation occurred in 35·5% of this series. Once again, the reported incidence of this complication varies widely, from 2% to 66%, but it is established that hydramnios is far more common amongst diabetic patients.

TABLE II.

The Incidence of Certain Diabetic Complications and the Associated Fœtal Mortality.

Complication.	Number of Cases.	Deaths.
Nephropathy	9	3
Hypertension	14	4
Retinopathy	2	0
Acidosis (severe) ..	2	1
Hypoglycæmic coma ..	2	0

Maternal Mortality.

There were two maternal deaths in this series, both occurring towards the end of 1953.

The first patient was aged 30 years, and had been diabetic for 19 years. She had evidence of nephropathy, neuropathy and retinopathy. During her fourth pregnancy she had been

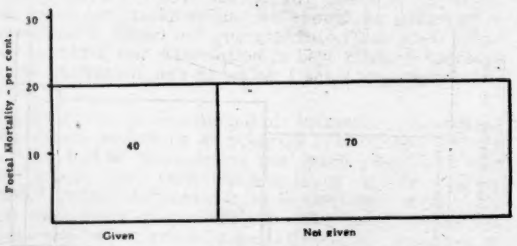

FIGURE VI.

fairly difficult to stabilize and spent a considerable time in hospital. She had a vaginal delivery at the thirty-ninth week and died 48 hours later. The clinical cause of death was given as cerebral thrombosis, and at autopsy the main finding was pulmonary œdema.

TABLE III.

Comparison of Results from the Combined Diabetic Ante-Natal Clinic (34 Patients, 39 Pregnancies with Viable Fœtuses).

Observation.	No Hormones Given. (22 Cases.)	Hormones Given. (17 Cases.)
Stillbirths	1	1
Neonatal deaths ..	3	2
Total fœtal deaths ..	4	3
Cases of toxæmia ..	13	14
Cases of hydramnios ..	10	10
Vaginal deliveries ..	7	7
Cæsarean sections ..	15	10
Diabetic control:		
Good	15	13
Fair	5	3
Bad	2	1

The other patient was 25 years old and had been diabetic for 14 years. She had no evidence of any diabetic complications. She was submitted to Cæsarean section at the thirty-seventh week and died 24 hours later in profound shock. At autopsy there were large hæmorrhages into both adrenal glands.

Fœtal Mortality.

Severity of the Diabetes.

Some authors have established a relationship between fœtal mortality and the severity of the diabetes (White, 1952; Pedersen, 1954), whilst others have not (Oakley and Peel, 1949; Oakley, 1953; Clayton, 1956; Hagbard, 1956).

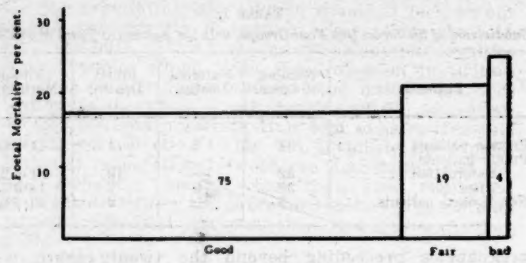

FIGURE VII.

The fœtal mortality for the present series was analysed in regard to the age at onset of the diabetes, its duration, and the pre-pregnancy insulin dosage (Figures I, II and III). In these diagrams the width of the blocks and the figures in the blocks indicate the number of cases in each group. There does not appear to be any definite correlation.

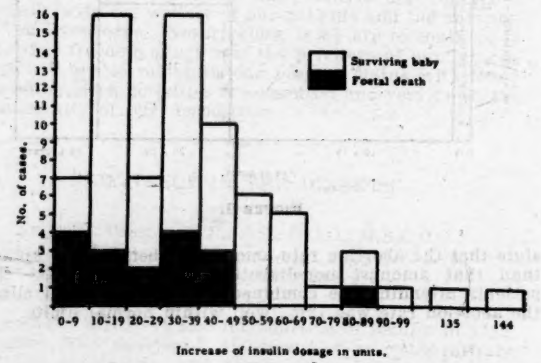

FIGURE VIII.

However, when the cases were grouped according to Priscilla White's classification, there appeared to be an increasing mortality with increasing severity of the disease (Figure IV).

White's classification (White, 1949) is based on the severity of the diabetes as judged by a combination of the age of onset, the duration and the presence of vascular disease. Class A refers to those patients whose diabetes is diagnosed by glucose tolerance tests and who do not require any insulin. Class B refers to those cases in which the onset was after the age of 20 years and the duration less than 10 years, and no vascular disease was present. Class C refers to those cases in which the onset of the diabetes was between the ages of 10 and 19 years, the duration was between 10 and 19 years, and there was minimal vascular disease. Class D refers to those cases in which the onset was before the age of 10 years, the duration was over 20 years, and more marked vascular disease was present. Class E refers to those patients with radiological evidence of calcification of the pelvic arteries. Class F refers to patients with nephropathy in diabetes of long standing. In some cases from the present series, difficulty was experienced in assigning them to a definite group, as not all

cases will fit readily into these classes. Information was not available to determine patients falling into class E.

Pre-eclamptic Toxæmia.

The fœtal mortality when the pregnancy was complicated by preeclampsia, in this series, was 23·3%, as against 16·1% for non-toxæmic patients (Figure V). However, it is generally believed that the fœtal mortality is higher in toxæmic patients who are not diabetic (Oakley and Peel, 1949). When it is considered that the incidence of toxæmia is so much higher in diabetic patients, the resultant fœtal mortality is more significant.

According to her, the lack of favourable results was due to the type of therapy employed, the quantities used and the route of administration, and to the fact that a standard programme of hormone therapy was used (in the English series) regardless of the severity of the diabetes or its clinical or chemical course.

In this series, 40 patients were given hormone therapy (Figure VI). Some of these patients were attending the combined diabetic ante-natal clinic and received stilbœstrol by mouth and progesterone by intramuscular injection in gradually increasing dosage up to 200 milligrammes of stilbœstrol per day, and 30 milligrammes of "Proluton"

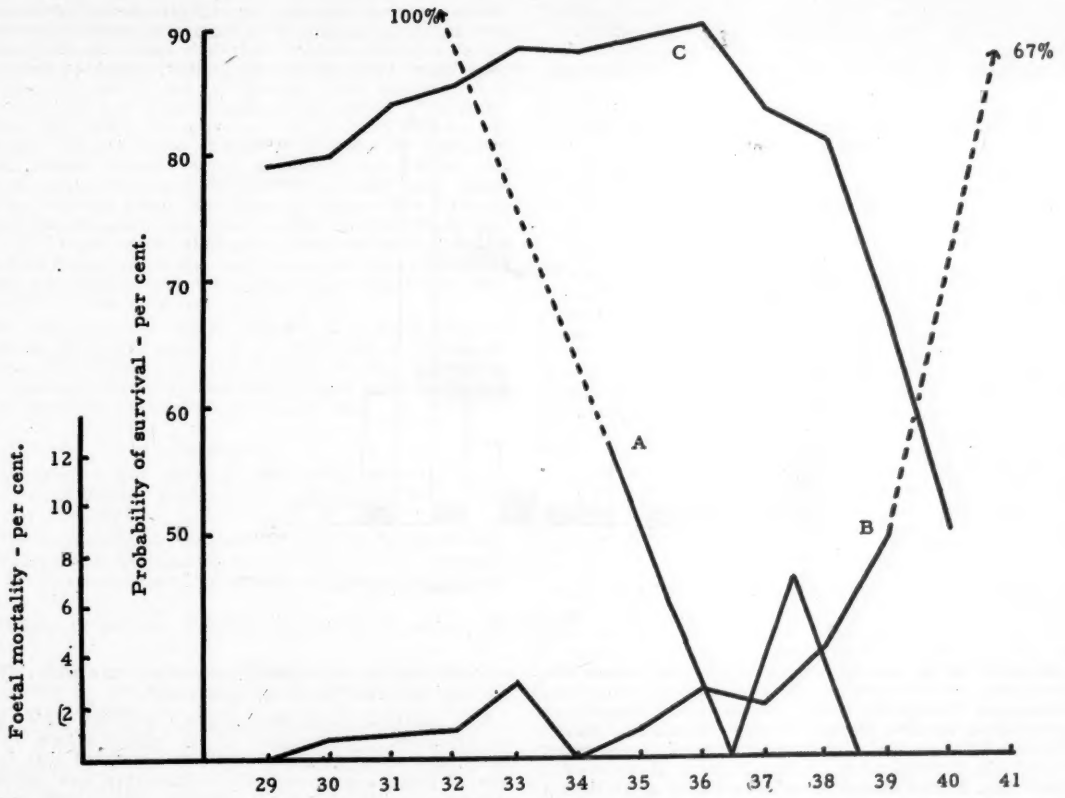

FIGURE IX.

Diabetic Complications.

There was a slightly increased fœtal mortality associated with the presence of diabetic complications. Twenty-one of the pregnancies were so complicated, with the loss of five babies. Table II gives the frequency of the various complications and the number of associated fœtal deaths.

Hormone Administration.

As a result of Priscilla White's work, there was considerable interest in the effects of administering œstrogen and progesterone in diabetic pregnancies. Priscilla White claimed that hormonal imbalance was the most important single factor causing the large fœtal loss in diabetic pregnancies. Since then, two large controlled series have been presented (Dieckmann *et alii*, 1953; Medical Research Council, 1955), in which no evidence could be found to support her claim. She has defended her claims (White *et alii*, 1956) by pointing out the differences between her cases and the series of the Medical Research Council. three times per week, according to a predetermined scale. The remainder of these patients were private patients and received varying amounts of hormone preparations of different types. The fœtal mortality was found to be uninfluenced by the administration of hormones.

It is interesting in this regard to examine the results obtained at the combined diabetic antenatal clinic, where all patients received the same standard of medical and obstetric care and were as far as possible treated alternately with a set schedule of hormone therapy or with non-active control tablets. Table III compares some of the results obtained. There is very little difference, if any, between the two groups.

Medical Control.

As would be expected, fœtal mortality increases when the medical control of the diabetes is poor. Many authors (Clayton, 1956; Pedersen and Brandstrup, 1956; Hagbard, 1956) emphasize the necessity for strict medical control,

with frequent supervision of insulin dosage and early admission to hospital for stabilization when required. In this series the cases have been arbitrarily classified as of good control when the diabetes was adequately controlled throughout pregnancy without admission to hospital, as of fair control when admission to hospital was required for stabilization, and as of poor control when the diabetes remained unstable even in hospital (Figure VII). The fœtal mortality rose progressively with the poorer degrees of control.

In this regard it is interesting to examine the relationship between fœtal mortality and the increase of insulin dosage during pregnancy (Figure VIII). It can be seen from this diagram that in the 74 cases in which the information was available, all but one of the fœtal deaths occurred in cases in which the insulin dosage was increased by less than 50 units per day throughout pregnancy. The fœtal mortality in the first five groups (insulin increase

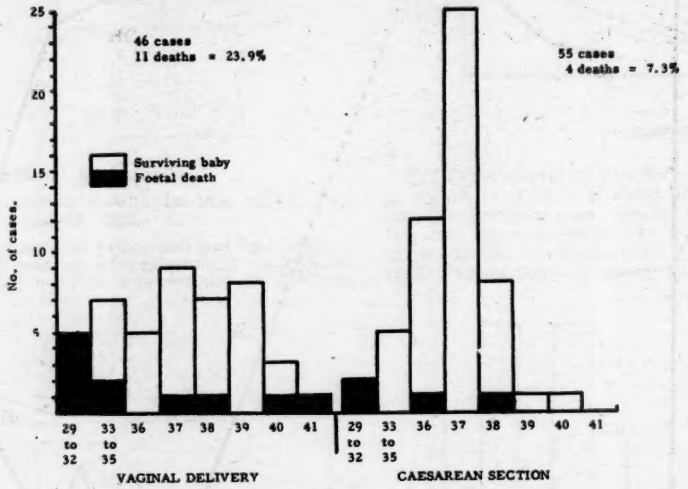

FIGURE X.

less than 50 units per day) was 28·6%, and when the increase was over 50 units per day the mortality was 5·5%. Pedersen and Brandstrup (1956) emphasize the importance of controlling insulin dosage by early admission of the patient to hospital and the maintenance of blood sugar levels within physiological limits. Perhaps these results indicate that insulin dosage should be rather more liberal during pregnancy.

Time of Delivery.

One of the greatest factors in the reduction of fœtal mortality has been the recognition of the fact that diabetic pregnancies should in most cases be terminated a few weeks before term. The risk of intrauterine death during the last few weeks of pregnancy is well known. Clayton (1956) mentions recent work in progress which indicates that the placenta in a diabetic pregnancy is mature at the thirty-sixth week and that thereafter it behaves like the "post-mature" placenta, giving rise to fœtal anoxia and fœtal deaths. Hagbard (1956), from the result of a statistical analysis of 467 pregnancies in Sweden, concluded that the most favourable time for delivery was at the beginning of the thirty-seventh week. Figure IX illustrates the effect of time of delivery on perinatal mortality. Graph A shows the risk of neonatal death for babies born during each week of pregnancy. Graph B shows the risk of a fœtus dying *in utero* during each week of pregnancy. Graph C shows the probability of survival, which gives the percentage of infants alive *in utero* at the beginning of each week who ultimately survive. Graphs A and B do not take into account those babies who die during the course of labour, whereas graph C does take into account these fœtal deaths. It can be seen that the probability of survival (graph C) gradually rises to the beginning of the thirty-seventh week and then falls abruptly. Hagbard states that delivery should take place earlier than the end of the thirty-sixth week in the presence of acidosis or hydramnios, or when toxæmia has occurred, provided that the fœtus appears to be at least five pounds in weight.

Mode of Delivery.

In considering the best mode of delivery in diabetic pregnancies, consideration must be given to two points—first what is safest for the mother, and second what is best for the child.

With regard to maternal mortality, several series quote fewer maternal deaths with Cæsarean section than with vaginal delivery (Oakley and Peel, 1949). In the present series there were 55 vaginal deliveries and 55 Cæsarean sections, making the Cæsarean section rate 50%. There was one maternal death in each group.

With regard to maternal morbidity, Hagbard (1956) made an extensive investigation of all complications in his series, and after statistical analysis concluded that in the case of *primiparæ* there was considerably less morbidity associated with abdominal delivery than with vaginal delivery, whilst the morbidity rate was approximately the same in the case of *multiparæ*. In addition, he found that in both *primiparæ* and *multiparæ* there was a significantly greater morbidity when vaginal delivery followed induction of labour than when labour was spontaneous in onset. It would appear, then, that in general Cæsarean section is as safe for the diabetic mother as vaginal delivery, if not safer.

With regard to the fœtus, many authors report a greater fœtal salvage in series with a high percentage of Cæsarean sections than in series with a low percentage (Oakley and Peel, 1949; White, 1952). Figure X shows the results in this series. In 55 abdominal deliveries the fœtal mortality was 7·3% and in 46 vaginal deliveries in which the fœtus was alive at the onset of labour, the fœtal mortality was 23·9%. Excluding cases delivered before the thirty-sixth week, the respective fœtal mortalities were 4·3% for Cæsarean section and 12·1% for vaginal delivery.

It would appear, then, that the best over-all results will be obtained by Cæsarean section, which should be the method of choice for all *primiparæ*, and for *multiparæ* when a quick and easy labour cannot be expected. Cæsarean section should also be performed on *multiparæ* who fail

to respond satisfactorily to induction of labour and, of course, if there are other indications for Cæsarean section.

Management.

There is no doubt that proper management of the pregnant diabetic will result in greater fœtal salvage. First and foremost, such patients should receive constant and individual care by the combined efforts of a physician experienced in diabetes and of an obstetrician experienced in dealing with these cases. During the first half of the pregnancy they should be examined at least monthly, and more frequently if this is necessary to control their diabetes adequately. During the second half of pregnancy they should be examined preferably at weekly intervals and be admitted to hospital as soon as the diabetes shows signs of instability and at the earliest sign of preeclampsia. Ideally, all diabetic patients should be in hospital from the thirty-second week onwards. Delivery should take place no later than the end of the thirty-sixth week, and earlier if there has been any preeclampsia, hydramnios or acidosis, provided that the fœtus appears to be at least five pounds in weight. All *primiparæ* should be delivered by Cæsarean section unless labour occurs spontaneously before the thirty-seventh week and progresses quickly and satisfactorily. If the fœtus has died *in utero*, then vaginal delivery can be allowed provided other factors are favourable. In the case of the *multipara*, Cæsarean section is the method of choice when she has had a previous abdominal delivery or when it does not appear that a quick and easy labour will follow induction.

After delivery the infant should be managed as a premature child and placed in an incubator if necessary, even though he might seem to be mature in size. Aspiration of the stomach should be carried out soon after birth and glucose given by mouth during the first day.

Summary.

1. A series of 110 diabetic pregnancies from the King George V Memorial Hospital, in which the fœtus was viable, is presented.

2. The incidences of preeclamptic toxæmia, hypertension, nephropathy and hydramnios are discussed. The incidence rates in this series are in accord with other published series.

3. Details of the two maternal deaths in the series are given.

4. The fœtal mortality is discussed in relationship to the severity of the diabetes, as indicated by the age of onset of the disease, its duration and the insulin dosage before pregnancy, and according to Priscilla White's classification, to preeclamptic toxæmia, the presence of diabetic complications, hormone administration, medical control of the diabetes and the time and mode of delivery.

5. The method of management of the diabetic pregnancy is discussed.

Acknowledgements.

I wish to thank the many members of the staff of the King George V Memorial Hospital, and of the Diabetic Unit, Royal Prince Alfred Hospital, who have been interested in this work. In particular I must thank Professor Bruce Mayes and Dr. Kempson Maddox, who were responsible for starting this work at King George V Hospital and who have been responsible for the care and management of all the patients attending the Combined Diabetic Antenatal Clinic, for their permission to publish these results.

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THE ACUTE DEFIBRINATION SYNDROME IN OBSTETRICS.

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UTERINE HÆMORRHAGE combining both ante-partum and post-partum hæmorrhage is one of the most common obstetric complications. Although the more severe hæmorrhages are not common, attention has been drawn in recent years to the dangerous, indeed often fatal, hæmorrhages which occur from the uterus when a state of hypofibrinogenæmia complicates the labour. The common post-partum hæmorrhage occurs in the third stage before the delivery of the placenta. The hæmostatic process, which normally occurs after the delivery of the placenta, is remarkable. Before delivery, some 500 millilitres of blood pass through the placenta each minute. The large raw area of the uterus at the placental site is capable of one of the quickest and most severe hæmorrhages encountered in medical practice. Normally, the blood loss from this area is a matter of a few ounces.

The generally recognized primary hæmostatic mechanism is the uterine tone. The force with which the different parts of the uterus contract is proportional to their radii of curvature before delivery. This, combined with the contractility of the myometrium, assures that maximum force will be exerted in the fundus of the uterus, the usual site of placental attachment. Thus the large vessels in the base of the placental site are occluded by the pressure of the surrounding muscle. The secondary means of hæmostasis is the clotting of blood in the placental sinuses. The enormous hæmorrhages which may occur when the clotting mechanism of the blood is defective emphasize the importance of this secondary mechanism.

In recent years attention has been drawn to the acute defibrination syndrome, which may occur in association with pregnancy and certain surgical operations. As a complication of pregnancy it has been described by Dieckmann (1936), Moloney, Egan and Gorman (1949), Weiner *et alii* (1950), Reid (1953) and many others. It may arise shortly before or during labour or abortion and may produce a fatal bleeding from the uterus or a generalized hæmorrhagic state. The blood is usually found to be incoagulable, and tests show the absence or the reduction of fibrinogen. This syndrome has been found to be associated with accidental hæmorrhage, intrauterine fœtal death, amniotic fluid embolism and toxæmia of pregnancy. A generalized bleeding tendency may also occur early in pregnancy as a sequel to criminal abortion.

Schneider (1951) suggested that, in these conditions, fragments of necrotic placental tissue become detached and enter the maternal circulation through the large, open blood spaces at the placental site. Such tissue fragments are known to be actively thromboplastic, and their entry into the blood stream would cause, paradoxically, incoagulability of the blood. This is believed to be due to slow

and generalized intravascular coagulation, which produces not thrombosis of large vessels, but the laying down of a thin layer of fibrin on the internal surface of the blood vascular system. This belief is consistent with early experimental work on blood coagulation. Pickering (1928) observed that incoagulability could be produced by the slow intravenous injection of thromboplastic tissue extracts or other substances capable of promoting clotting, such as cream, chloroform, serum or silica.

Hartman, Conley and Krevans (1951) observed that intravenous injection of large amounts of brain thromboplastin in dogs caused rapid death from widespread thrombosis, but the injection of smaller amounts caused decreased coagulability of the blood with fibrinopenia, thrombocytopenia and prolongation of prothrombin time. Schneider (1951) found that in some cases of defibrination of the blood there was active fibrinolysis, which may be considered to be the cause of the defibrination.

It has long been known that the blood of human beings and animals after sudden death can often be shown to be capable of liquefying fibrin. There are many observations to suggest that any form of stress, mental as well as physical, can induce fibrinolytic activity in the blood of living human subjects. Macfarlane (1937) observed increased fibrinolytic activity in human patients after surgical operations. However, Macfarlane and Biggs (1946) found that the fibrinolytic activity was more related to the mental distress of the patient than to surgical trauma in operation cases; following this up, Biggs, Macfarlane and Pilling (1947) showed that severe exercise or the injection of adrenaline had similar effects. Several of these factors may operate during labour, particularly during a prolonged labour. However, Astrup (1956) suggested that when fibrinolysis is present it may be due to activators of the fibrinolytic system derived from placental tissue or amniotic fluid.

Diagnosis.

The occurrence of profuse or uncontrollable bleeding in any of the obstetric complications mentioned should at once suggest the possibility of acute defibrination. In two of the cases reported in this paper the preeclamptic toxæmia was of mild to moderate degree, so it was possible for the diagnosis of acute defibrination to be overlooked. In all cases reported in this paper the diagnosis was not established for some hours after the onset of the condition.

In spite of the presence of some of the known ætiological conditions for acute defibrination it is usual to look first for the common causes of uterine hæmorrhage. However, in all cases of severe uterine hæmorrhage a sample of venous blood should be taken to check whether coagulation occurs and to observe the resulting clot; blood coming from the vagina is not satisfactory for this purpose. In the early stages bleeding may be limited to the areas of trauma (placental site, episiotomy, sites of injections), but as the condition progresses, generalized bleeding in the form of ecchymoses, bruising and spontaneous hæmorrhages from mucous membranes of the mouth, bladder and bowel may develop.

The simplest method of establishing the diagnosis is to observe a sample of whole blood. In fibrinopenia, clotting does not occur. If a very frail clot forms, or if the clot appears then dissolves within an hour, the fibrinogen content of the plasma can be assumed to be below 100 milligrammes per 100 millilitres.

Another readily available test is the semiquantitative test for fibrinogen employing thrombin, as described by Bonsnes and Sweeney (1955). This test will give a definitive answer when the fibrinogen levels are normal and when there is an actual absence of fibrinogen or afibrinogenæmia. This test depends, in principle, on the fact that thrombin, added to plasma, rapidly converts fibrinogen to fibrin, and that the time at which clotting starts and the type of clot formed are going to be a function of the concentration of fibrinogen in the plasma. This test is commonly known as the "Fibrindex Test". The procedure is as follows.

Test Procedure.

1. Collect a sample of venous blood in a bottle containing Wintrobe mixed oxalate (two milligrammes per millilitre).

2. Centrifuge the blood in a clean, dry centrifuge tube to obtain the plasma.

3. Whilst the blood is being centrifuged, dissolve the proper amount of thrombin in saline (50 N.I.H. units per millilitre of physiological saline).

4. To test for fibrinogen, add 0·2 millilitre (or a drop from a medicine dropper) of plasma to the bottom of a small test tube.

5. Add 0·2 millilitre (or a drop from a medicine dropper) of the thrombin solution directly on the plasma, starting a stop-watch at the same time.

6. Immediately mix the two together by shaking for less than two seconds.

7. Then, starting with the tube held at an angle of approximately 45° in such a way that the bottom of the tube acts as a pivot, move the other end up and down in the same vertical plane, through an arc of about 45°, at the rate of about one oscillation each two seconds, so that whilst the content of the tube remains fluid it flows back and forth along the bottom side of the tube for a distance of about one centimetre.

8. The time clotting starts (if it does) is noted.

9. The oscillation of the tube is continued until one is sure that the clot formation has definitely started and that it is going to continue.

10. The tube is then held in a vertical position and examined at 30, 45 and 60 seconds by similar single oscillations as were employed earlier.

11. If clotting has not started by 60 seconds, observations are stopped.

A control test should be carried out at the same time.

Biggs and Macfarlane (1957) describe a simple quantitative test for fibrinogen using an equal volume of half-saturated ammonium sulphate to the patient's plasma and the volume of the precipitate is measured in a special graduated tube after centrifuging and comparing the result with a normal control test. However, this test is much more time-consuming.

Treatment.

The average fibrinogen content of plasma is 250 milligrammes per 100 millilitres (a range of 190 to 330 milligrammes). The plasma fibrinogen level is raised in pregnancy and the mean normal fibrinogen level at term is about 300 milligrammes per 100 millilitres (Hodgkinson, Margulis and Luzadre, 1954). The minimum blood fibrinogen level required for effective clotting is not easy to define; it has been suggested that the critical fibrinogen level is 90 to 100 milligrammes per 100 millilitres of plasma (Weiner *et alii*, 1953), so that the rational treatment is to aim at the restoration of a level higher than this.

Biggs and Macfarlane (1957) state that for this purpose four to five pints of whole blood should, theoretically, be sufficient. However, in practice, the administration of up to even 10 litres of whole stored blood has failed to control the hæmorrhage. The administration of fibrinogen is effective, and apparently good results have been found with relatively small amounts. One case in this series responded to two grammes of fibrinogen, and the maximum amount given was four grammes. Moore (1954) found that two grammes of fibrinogen may be sufficient.

In determining the amount of fibrinogen to be administered, consideration should be given to the patient's plasma volume; the average volume is approximately 45 to 47 millilitres per kilogramme. In pregnancy there is an increase in the plasma volume of from 25% to 30%, so

that the average plasma volume at term will range from 3000 millilitres to 4500 millilitres in a patient weighing between nine stone and eleven stone. In these patients four grammes of fibrinogen will raise the plasma fibrinogen level to over the critical level of 100 milligrammes per 100 millilitres of plasma, even assuming that there is a complete absence of fibrinogen. In many cases less than this amount will be required.

As fibrinogen dissolves with difficulty, a blood sample should be tested for coagulation after the administration of each gramme, whilst an assistant is preparing the next gramme. In all of the cases reported in this series it was found unnecessary to give more fibrinogen after the clotting defect had been corrected. Heaton (1955) advocated the administration of three grammes by slow intravenous infusion after the clotting power of the blood had been restored.

Walsh (1957) has drawn attention to the danger of fibrinogen containing the virus of infectious hepatitis. It will be for the clinician to decide in each case between the risks of uncontrolled hæmorrhage or possible subsequent liver damage. This latter danger must mean that the minimum quantity of fibrinogen should be given to correct the hæmorrhagic state. It is unlikely that the diagnosis of acute defibrination will be established before the patient is in a shocked state, and a blood transfusion will usually be necessary to relieve the state of shock. Indeed, in most cases, a transfusion of either albumin or blood will be given before the coagulation defect is discovered. The amount of whole blood given should be sufficient to correct the state of shock.

Patients in whom there is retention of a dead fœtus should be warned to report any bleeding, however trivial. The clotting time or fibrinogen concentration should be determined in the third week after fœtal death, and should be repeated thereafter at weekly intervals, even in the absence of hæmorrhagic symptoms. If fœtal death is diagnosed for the first time when the patient is in labour, the clotting time of venous blood should be estimated. This also applies in cases of suspected criminal abortion, particularly if bleeding is the dominant symptom.

If hypofibrinogenæmia appears, the defect should be corrected by intravenous administration of up to four grammes of fibrinogen, repeated as often as necessary. Whilst the concentration of fibrinogen is sustained in this way, the uterus should be emptied as soon as possible. The hypofibrinogenæmia is corrected by emptying the uterus.

Nine patients have been treated for acute defibrination at this hospital during the past three years. The following is a summary of four of these cases.

CASE I.—The patient was pregnant for the fourth time and had had two previous normal pregnancies and one miscarriage. The present pregnancy, a twin pregnancy, was complicated by a mild degree of toxæmia. Her blood group was O Rh positive. Her blood pressure rose from 130/70 millimetres of mercury at 12 weeks to 160/90 millimetres of mercury at 35 weeks. Her total weight gain over the same period was 28·5 pounds. The patient was admitted to hospital for bed rest, for toxæmia and twin pregnancy, and 21 days later she came into spontaneous labour. Thirty minutes after the contractions started she began to bleed from the vagina. At the onset of labour her blood pressure was 155/90 millimetres of mercury. Fifty minutes after the onset of the bleeding the first twin was delivered; 10 minutes later the second twin was delivered, and after a further five minutes the placenta was delivered. The placenta weighed 22 ounces and there was no evidence of retro-placental blood clot. It was noticed that there was excessive bleeding from the episiotomy wound and from the uterus after the delivery. Two hours after the onset of labour the maternal pulse rate was 120 per minute and her blood pressure was 95/60 millimetres of mercury. Blood taken from a vein for cross-matching purposes failed to clot within 10 minutes. The patient's plasma was then tested by Bonsnes's method for the estimation of fibrinogen. No clot occurred within five minutes, although a control sample clotted within 30 seconds. The patient was given an intravenous infusion of four grammes of fibrinogen in 200 millilitres of normal saline, and a blood transfusion was commenced. Thirty minutes later a blood sample clotted within three minutes. After 1500 millilitres of whole blood had been transfused, the pulse rate slowed and the blood pressure rose. On the third post-natal day the patient's hæmoglobin value was 8·5 grammes per 100 millilitres.

CASE II.—The patient was pregnant for the fifth time, having borne four children previously. Her blood group was A Rh positive. The patient gave a history of antepartum hæmorrhages in each of her four labours. As far as could be ascertained, the hæmorrhages were all so-called accidental hæmorrhages and required blood transfusions on each occasion. The present pregnancy, a twin pregnancy, had been uneventful. The patient was admitted to hospital on March 15, 1956, at 11 p.m., when 34 weeks pregnant, with lower abdominal pain and in a very shocked state. Her blood pressure and pulse rate could not be recorded. She was given 200 millilitres of human albumin followed by 500 millilitres of dextrose and 500 millilitres of blood. Her pulse rate was 112 per minute; blood pressure was 150/60 millimetres of mercury. A vaginal examination was performed at 11.45 p.m. The membranes of the presenting sac were ruptured and blood-stained liquor drained. A further 1500 millilitres of blood were then given. The patient continued to bleed from the vagina. At 3.10 a.m. it was observed that blood taken from a vein had not clotted within 30 minutes. At 4.15 a.m. Bonsnes's test for fibrinogen was carried out, and no clot formed within 60 seconds. The control sample clotted in 20 seconds. At 4.22 a.m. one gramme of fibrinogen was given by intravenous infusion. A blood sample taken at 4.29 a.m. clotted in six minutes. At 4.39 a.m. a second gramme of fibrinogen was given by intravenous infusion, and a third gramme at 4.44 a.m. A blood sample was taken which clotted in three minutes. At 4.50 a.m. a fourth gramme of fibrinogen was given. A blood sample taken at 4.53 a.m. clotted in three minutes, and a further 500 millilitres of whole blood was given.

At 6.20 a.m. there was normal delivery of twin male infants. The first baby was stillborn; the second baby was alive. The blood loss at delivery was 29 ounces. The first placenta showed evidence of almost complete separation by a large amount of retro-placental clot. The second placenta was normal. After delivery the clotting time was four minutes and the maternal pulse rate was 90 per minute. On the second post-natal day the patient's hæmoglobin value was 8·0 grammes per 100 millilitres, and a further litre of whole blood was given. The hæmoglobin value was 10·2 grammes per 100 millilitres on the fifth post-natal day.

CASE III.—The patient was pregnant for the fifth time and had borne three children. There was a previous history of a blood transfusion for post-partum hæmorrhage four years ago. The patient was 16 weeks pregnant and had induced an abortion by douching herself with soapy water. She passed the fœtus before her admission to hospital. On admission to hospital her pulse rate was found to be 120 per minute and her blood pressure was 90/60 millimetres of mercury. One hundred millilitres of serum albumin were given intravenously, followed by 500 millilitres of normal saline and by a blood transfusion. With the blood transfusion and nor-adrenaline infusion running, the patient was transferred to the operating theatre and curettage was performed. The vaginal bleeding continued after the curettage, so the uterus was packed with gauze and a blood sample taken. The blood did not clot.

Bonsnes's test for fibrinogen showed no clot formation, whereas a control sample clotted in 20 seconds. One gramme of fibrinogen was given intravenously; five minutes later a blood sample was taken which did not clot. A second gramme of fibrinogen was given intravenously, and a blood sample taken five minutes later clotted after 10 minutes. A third gramme of fibrinogen was then given, and a blood sample taken 10 minutes later clotted in seven minutes. A fourth gramme of fibrinogen was given, and a blood sample taken five minutes later clotted in five minutes. Plasma was tested for fibrinogen by Bonsnes's method after the third gramme of fibrinogen. This showed clot formation in 60 seconds.

The patient died 36 hours later as a result of a septic abortion. The cervical swab taken at the time of her admission to hospital grew *Bacillus coli* and *Clostridium welchii*.

CASE IV.—The patient was pregnant for the fourth time, having borne three children. She had received her antenatal care and was confined at another hospital. She had toxæmia of moderate severity and came into spontaneous labour at term; labour lasted 12 hours, and at 3.20 p.m. on October 9, 1956, she had a normal confinement. About one hour later it was noticed that the patient was in a state of collapse and that she was continuing to lose large amounts of blood from the vagina. Her blood pressure was then 60/40 millimetres of mercury.

At 8.30 p.m. the patient was transferred to The Women's Hospital, Crown Street, Sydney. On her admission, small hæmorrhages were noted at the sites of venepunctures or injections. A blood sample was taken which clotted within five minutes, but the clot broke up on standing. Intravenous infusion of serum albumin was then commenced. At 9.15 p.m. the cervix and uterus were explored under general anæsthesia, and the bleeding was found to be coming from the uterus. A blood transfusion was then commenced. At 12.5 a.m. (October 10) a venous blood sample was taken which clotted in seven minutes, but there was poor clot formation and the clot readily disintegrated. Normal control blood clotted in two minutes and the clot was strong. At 12.30 a.m. Bonsnes's test for fibrinogen was performed. Very little clotting took place after two minutes, whereas control plasma clotted in 35 seconds. At 1.7 a.m. an intravenous infusion of one gramme of fibrinogen was given. A blood specimen taken at 1.12 a.m. took seven minutes to clot. A fibrinogen test was performed and clotting occurred in 70 seconds. A second gramme of fibrinogen was given intravenously at 1.18 a.m., and a blood specimen taken at 1.24 a.m. clotted in five minutes. A fibrinogen test was performed and clotting occurred in 50 seconds, as did the control plasma. At 1.35 a.m. a third gramme of fibrinogen was given intravenously, and a blood specimen taken at 1.40 a.m. clotted within three minutes. A fibrinogen test resulted in clotting within 35 seconds. At 2.30 a.m. there was only slight loss of blood from the vagina. The patient was given a total of 2·5 litres of blood. On the third post-natal day her hæmoglobin value was 9·2 grammes per 100 millilitres.

Summary.

1. Consideration has been given to the ætiological causes of the acute defibrination syndrome in obstetrics. Five conditions are commonly associated with this condition: (i) intrauterine fœtal death with retention of the fœtus; (ii) amniotic fluid embolism; (iii) accidental hæmorrhage; (iv) toxæmia of pregnancy; and (v) criminal abortion. Four reports of cases have been presented to illustrate the syndrome occurring in the three last-mentioned conditions.

2. Mention has been made of the simplest methods of diagnosing the condition, namely, the clot observation test and the "Fibrindex Test".

3. The dose of fibrinogen given to correct the condition has been discussed. The administration of up to four grammes of fibrinogen has been recommended and theoretical considerations advanced for the advocacy of this amount.

4. This risk of hepatitis occurring as a *sequela* to the administration of fibrinogen has been mentioned.

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ANTI-s OF THE MNSs BLOOD GROUP SYSTEM.

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FOR a period of 20 years following the announcement in 1927 of the MN blood groups by Landsteiner and Levine (1927, 1928), this system was thought of in terms of a single pair of allelomorphic genes, each of which was capable of independent expression in the red cell antigens M and N.

An extension was made to the MN groups following the discovery by Walsh and Montgomery (1947) of an antibody shown to be unrelated to the blood groups A B O, Rh, P, Lutheran, Kell and Lewis. It was shown by Sanger and Race (1947) that this antibody was in fact recognizing an antigen within the MN system. The antibody was named anti-S, and the corresponding antigen, S.

Race and Sanger (1954), quoting an earlier publication, state:

> A reasonable genetical interpretation of the results is that there are four allelomorphs at the locus responsible for those groups, *Ms*, *MS*, *Ns* and *NS*, the mutation of *S* being a change which can happen both to *M* and to *N* genes, and which makes the resulting red cells agglutinable by the new antibody. On the other hand, it is possible that *S* is a separate gene, presumably having an allelomorph *s*. If an antibody corresponding to *s* were found, as we confidently anticipate, this interpretation of a separate pair of genes would seem the more probable. The situation would then be very similar to that of the *C*, *D* and *E* antigens of the Rh system. Anti-*s* will agglutinate about 88% of English bloods. If the interpretation of linked genes is correct, that linkage must be very close, possibly absolute, otherwise crossing-over would presumably have resulted in an equilibrium in which the ratio of *MS* to *Ms* would be equal to that of *NS* to *Ns*.

The predicted anti-s was discovered by Levine, Kuhmichel, Wigod and Koch (1951). Since 1947, a number of examples of anti-S have been found and have been shown to be the cause of hæmolytic disease of the newborn and of hæmolytic transfusion reactions. It has also been found in the serum of individuals in whom it has apparently occurred naturally, that is, without known stimulus. A second Australian example of anti-S has recently been found by Verso and Simmons (1958), ten

TABLE I.
The Blood Groups of Mrs. A., Baby A. and Donor Y.

Blood Tested.	Blood Groups.								
	ABO.	MNS.	Rh.	P.	K.	Le^a.	Lu^a.	Fy^a.	Jk^a.
Mrs. A...	A	MS	rh	+	−	−	−	+	+
Baby A.	A	MS	rh	+	−	−	−	..	..
Donor Y	O	MS	Rh_1rh	+	−	−	−	+	+

years after the original antiserum found by Walsh and Montgomery (*loco citato*).

The first anti-s, discovered by Levine *et alii* (*loco citato*), occurred as a monovalent atypical antibody in the serum of the mother of a child suffering from hæmolytic disease of the newborn, for which the antibody was unquestionably responsible. The second example was described by Sanger, Race, Rosenfield and Vogel (1953) and occurred in the serum of a volunteer who had been immunized with the object of producing anti-C, anti-D and anti-E in the one serum. These antibodies were in fact produced, but the unwanted additions anti-s and anti-Jk^b appeared also. Anti-s was identified in this serum by the indirect Coombs technique. The third example of anti-s was reported by Fudenberg and Allen (1957) and occurred in Boston, United States of America; it was detected as the result of a blood compatibility problem in a patient who had previously received three blood transfusions. Once again this antibody was revealed and identified by the indirect Coombs method.

The purpose of the present paper is to report an Australian example of anti-s and to describe briefly some of its properties, the circumstances under which it was detected and the manner of its identification.

HISTORY OF A CASE.

The patient, Mrs. A., aged 38 years, was found to be group A Rh negative. In April, 1957, she was delivered of a full-term male child at the Brisbane Women's Hospital. At birth the cord hæmoglobin value was 18 grammes per 100 millilitres of blood, and the child appeared normal. Subsequent progress was uneventful. This was the mother's fifth pregnancy. Her third child was born in 1949, and in 1952 she had a miscarriage after which she had a transfusion with one donation of blood. The transfusion took place at a small country hospital, and unfortunately this donor cannot be traced.

As she was Rh-negative, and since no tests had been performed during the present pregnancy for the presence of atypical antibodies in her serum, a routine examination was carried out on the day after delivery. It was found that at 37° C., her serum reacted avidly against both glucose-citrate and albumin suspensions of all five blood samples used in the test. These had been selected to include the antigens O, Cc, D(d), Ee, MN, Ss, P. Kk, Le^a, Le^b, Lu^a, Lu^b, Fy^a, Fy^b, Jk^a and Jk^b. In addition, the cells of the child were agglutinated to a degree comparable with the test cells. Direct Coombs tests performed on the cells of both the mother and the child gave negative results. Further tests showed that the reactions of the mother's serum against cells suspended in glucose-citrate were stronger at room temperature (21° C.) than at 37° C. A more extensive investigation was then undertaken in an effort to identify the antibody, or antibodies, responsible for the observed reactions.

MATERIALS AND METHODS.

A specimen of venous blood was collected from the mother and a further small sample was obtained from the child by heel-prick. A panel of 20 blood samples was selected to provide a cover of known blood group antigens. Ten to fifteen *per centum* unwashed suspensions of these erythrocytes and those from the child were prepared in Rous and Turner (glucose-citrate) solution as modified by Simmons, Graydon, Semple and Taylor (1951). One drop of serum was mixed with one drop of cell suspension on a glass slide and incubated at approximately 21° C. in a covered dish containing a moistened filter pad. The cells were examined macroscopically for agglutination at intervals of one-half and one hour while the slide was gently tilted over an illuminated viewing box. All cell suspensions except one (that of donor Y) were strongly agglutinated, although some variations in strength could be observed, indicating a possible dosage effect. Blood samples from the selected panel of 20 were then collected into 30% bovine albumin (10% to 15% suspensions) and tested on slides at 37° C. against the mother's serum. All cell samples, including those from donor Y, showed intense agglutination, which indicated the presence of an incomplete antibody possibly due to a second antigen.

RESULTS AND DISCUSSION.

The blood groups of Mrs. A., baby A. and donor Y were determined. The results of these tests are given in Table I.

TABLE II.
Anti-s and Anti-Rh_0(D) Titres in Mrs. A.'s Serum.

Cell Suspending Medium.	Test Cells.	21° C. Diluent. Saline.	21° C. Diluent. Normal A B Serum.	37° C. Diluent. Saline.	37° C. Diluent. Normal A B Serum.
Glucose - citrate solution.	R^1r *SS*	−	−	−	−
	R^1r *Ss*	4	..	4	..
	rr ss	8	..	4	..
30% bovine albumin.	R^1r *SS*	..	..	..	64
	R^1r *Ss*	..	..	..	64
	rr ss	..	32	..	32

A series of 350 random group O and group A blood samples suspended in glucose-citrate were then tested on slides versus Mrs. A.'s serum and read after one hour at room temperature. The results obtained are as follows. Of the 350 blood samples tested, 325 were agglutinated, while 25 gave negative reactions. Of the 25, all were found to be S-positive, while 24 of the 25 were M-positive.

The proportion of S-positive individuals among a sample of white Australians may be taken to be about 50% to 55% (Walsh and Montgomery, *loco citato*; Simmons and Graydon, 1950), so that among 25 individuals it would be expected that 13 would be S-positive. The difference between the observed number, 25, and the expected number, 13, is highly significant ($\chi^2 = 23$ for one degree of freedom).

Identification of Anti-s.

One of us (R.T.S.) was able to test a series of blood samples with a specimen of the original anti-s (GUTH) serum discovered by Levine *et alii* and generously sent to Melbourne in 1952. Thirty blood samples were selected, of which 16 were found to react with both serum GUTH (by indirect Coombs method) and serum Mrs. A. The other 14 samples gave negative reactions to tests with both sera. This correspondence may be shown thus:

		Reactions with known anti-s (GUTH) serum.	
		+	−
Reactions with serum Mrs. A.	+	16	0
	−	0	14

The probability that this association between the two types of serum anti-s and Mrs. A. is due to chance is less than $1/10^8$. In addition, 56 selected S-negative blood samples were tested with serum Mrs. A., and all gave strong positive reactions at room temperature.

Identification of Incomplete Anti-Rh$_0$(Anti-D).

With the agglutinating antibody in Mrs. A.'s serum identified as anti-s, the identification of the incomplete antibody was then attempted. Blood samples were not available from all of the 25 individuals who were negative with Mrs. A.'s serum; however, 18 samples were collected in 30% bovine albumin (10% to 15% suspension) and tested against Mrs. A.'s serum on slides at 37° C., readings being made after incubation for one hour. The results obtained in terms of Rh status are as follows:

		Blood Samples. Rh-positive.	Rh-negative.
Reactions with serum Mrs. A.	+	14	0
	−	0	4

The probability that this association with anti-Rh$_0$ (anti-D) is due to chance is less than 1/3000.

Tests for Other Rh Antibodies.

Following absorption of the anti-s antibody from the serum using *rr*(*cde/cde*) cells, which reacted strongly at room temperature, further tests were carried out with rh'(C) and rh"(E) blood samples suspended in bovine albumin. After incubation for two hours at 37° C., no reactions were observed, and it was concluded that neither anti-C nor anti-E was present.

Other tests at 5° C. revealed a small amount of cold agglutinin, which also reacted with the patient's own cells.

Antibody Titres.

The respective titrations are shown in Table II. Anti-s reacts slightly better at 21° C. than at 37° C. *Ss* blood samples suspended in glucose-citrate solution gave a titre of four, while homozygous *ss* gave a titre of eight. An Rh-negative *ss* sample suspended in 30% bovine albumin versus Mrs. A.'s serum diluted with normal A B serum (Henry and Simmons, 1946) gave a titre of 32 at both 21° C. and 37° C. Indirect Coombs titres for anti-s not shown in the table were approximately the same as those found with serum-albumin. Again, there was evidence that ss cells reacted more strongly than Ss cells.

A serum-albumin titration with Rh-positive cells gave an anti-Rh$_0$ (anti-D) titre of 64.

The s Antigen in White Australians.

A total of 681 random blood samples of groups O and A suspended in glucose-citrate were tested on slides at 21° C. versus Mrs. A.'s serum. Of these, 624 (91·6%) were positive and 57 (8·4%) were negative. If the 57 non-reactors are regarded as homozygous *SS*, then it may be calculated that the gene frequency of $S = 0{\cdot}29$ and $s = 0{\cdot}71$. These frequencies are identical with those reported by Simmons and Graydon (1950) for 220 white Australians tested with anti-S. The percentage of reactors with anti-S in the series was 48·2.

Of 625 bloods tested by one of us (J.A.A.), 51 were found to give negative reactions and, of these, 47 were found to be M-positive. If the other four are regarded as being of type NSNS, it may be calculated that the frequency of $NS = 0{\cdot}08$, a figure in agreement with that obtained by Fisher (quoted by Race and Sanger, 1954) on analysis of MNS results on 1419 blood samples tested in England and Australia.

Origin of the Anti-s Agglutinin.

The question which arises when any rare antibody is found is whether it has arisen as the result of known stimulus either by pregnancy or by blood transfusion, or by injection of blood, in which case it is called "immune", or whether the antibody just happens to be present without known stimulus, in which case it is referred to as a "natural" antibody.

In the present case we have as atypical antibodies anti-s agglutinating (titre four) and incomplete (titre 32), and anti-Rh$_0$(D) incomplete (titre 64). This was the patient's fifth pregnancy and the Rh-negative child born showed no evidence of hæmolytic disease. As the child's cells reacted with both anti-S and anti-s it must be heterozygous *Ss*, and although the father was not available for testing, it follows that he must also possess s either in single or in double dose. Mrs. A. had received a blood transfusion at a country hospital in 1952 after a miscarriage. At this stage of our considerations it was not possible to hazard a guess as to the origin of the anti-s in the patient's serum.

However, a search of the Blood Transfusion Service records in Brisbane revealed that Mrs. A.'s serum had been tested for atypical antibodies in 1949, and that anti-Rh$_0$(D) incomplete antibodies had been found. No other agglutinating antibodies had been detected by a careful observer. Had anti-s been present at that time, it was not detected either at room temperature or at 37° C., in tests made for the presence of agglutinating antibodies, or in tests made for the confirmation of her blood group as A, by the failure of group A and group O cells to agglutinate when mixed with her serum. It seems reasonable, therefore, to conclude that the anti-s, like the anti-Rh$_0$(D), is of immune origin.

Other Comments.

The anti-s serum has been freeze-dried and has been distributed to a number of Australian and overseas laboratories. The reports received have been satisfactory, especially when the serum has been used according to the technique described in this paper. One comment of interest is: "We received the dried anti-s about 10 days ago and tested it according to your directions with most excellent results. We found, however, that it works with whole ACD blood diluted with its own plasma to 15% but did not work with a 15% ACD solution of washed cells." The key word here probably is "washed". In Australia, blood samples for use in the detection of antibodies are often collected from the individual directly into Rous and Turner (glucose-citrate) solution, and the cells are not washed in saline. Red cells from fresh samples of clotted blood may also be transferred directly to glucose-citrate solution for A B O, MN, and Rh testing. Only when clotted blood is several days old, or when hæmolysis is evident, is washing of the red cells found to be necessary. The suspension of red cells in glucose-citrate is much more sensitive in agglutination tests than cells suspended in physiological saline. Slide tests for agglutination should be made with not less than 10% to 15% cell suspensions. Our respective laboratories have a good record for the detection of agglutinating antibodies suitable for the determination of most of the known blood groups. For example, Simmons (1956) recorded that eight of eleven anti-Kell sera showed slide agglutination (the remaining three were detected by the indirect Coombs test), and had been found in six different laboratories using the same slide technique. This is in conformity with the results from the Petri dish technique of Race and Sanger, referred to by van Loghem, Dorfmeier and van der Hart (1957), who state: "The very large surface allows much stronger agglutination reactions than in tubes." Several of these anti-K sera have been used as routine slide testing reagents. It is recommended that glucose-citrate solution be used instead of saline as a cell suspending medium for blood group agglutination tests, and that the red cells should not be washed unless they are old or showing hæmolysis.

SUMMARY.

An example of anti-s of the MNSs blood group system has been found. It is considered that it is of "immune" origin, that is, the patient was immunized by the s antigen either by pregnancy or blood transfusion, because the antibody was not detected in the patient's serum eight years earlier.

The methods used for the identification of anti-s have been described.

In white Australians, 624 (91·6%) of 681 blood samples were anti-s positive. The gene frequencies are $S = 0{\cdot}29$ and $s = 0{\cdot}71$.

It is recommended that in testing for agglutinating antibodies or in blood group testing, the cell suspending medium should be Rous and Turner (glucose-citrate) solution instead of physiological saline, and that the red cells should not be washed in saline unless otherwise indicated.

ACKNOWLEDGEMENTS.

The authors acknowledge with gratitude the fine cooperation received from Dr. D. B. Wherrett, of Kilcoy, Queensland. We are also grateful to Mrs. A. for her generous blood donations, which have enabled wide distribution of her serum, both in Australia and overseas.

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THE EFFECT OF ORAL ADMINISTRATION OF HYDROGEN PEROXIDE ON NEOPLASTIC GROWTH (EXPERIMENTAL SCREENING STUDIES).

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In a recent report (Holman, 1957), the oral administration of hydrogen peroxide to rats with Walker 256 adenocarcinoma tumours was stated to result in an average rate of cure of 50% to 60% of tumours, complete disappearance of the growth occurring in 15 to 60 days. Of four human patients with very advanced inoperable tumours who had been treated with hydrogen peroxide taken orally, two showed marked clinical improvement with decrease in the size of liver metastases and progressive diminution in the polysaccharide content of the blood serum (Keyser test).

Whilst hydrogen peroxide and peroxide-forming compounds have been used previously in attempts to modify neoplastic growths in experimental animals and humans (Turner, 1953; Makimo and Tanaka, 1953; Hollcroft and Lorenz, 1952; Worrall, 1956), Holman (1957) considers that continuous administration of the active oxidizing agent is necessary. The therapeutic ratio is based on a reputed difference in the enzyme pattern of malignant and normal cells. In normal cells under aerobic conditions, the enzyme catalase rapidly releases oxygen from the peroxide radical and prevents its toxic actions. Deficiency of this enzyme in malignant cells is considered to render them particularly sensitive to over-oxidation, if the supply of powerful oxidizing radicals (HO_2HO) can be maintained at a high level in tissues.

In view of the importance of investigating this claim further before submitting human cancer patients to wide-scale therapy with hydrogen peroxide, screening experiments were performed with mice inoculated with Ehrlich's ascites tumour, and the effects of hydrogen peroxide administration on rats with tumours induced by ionizing radiations and a chemical carcinogen were observed. The results of these experiments are reported in this paper.

Material and Methods.

Animal Material.

Hybrid mice (Walter and Eliza Hall Institute stock) and a C_3H strain were used for quantitative studies, and inbred Canberra Black stock rats were used for other experiments. The animals were housed in metal cages (five mice per cage, or one rat per cage) in an air-conditioned environment maintained at a constant temperature of 70° (±2°) F. Animals were fed on a balanced cube diet, with added fats for rats and carrot for mice. Fluid requirements were administered from standard drip bottles, which were graduated to permit measurement of the daily fluid intake of animals in each cage.

Transplantable Tumour.

The tumour used in the screening experiments with mice was a hyperdiploid line ELD of Ehrlich's ascites tumour (Lettré) with a 46 chromosome mode, originally obtained from Dr. E. L. French, Walter and Eliza Hall Institute, Melbourne, and it was passaged and maintained in C_3H mice in our laboratories.

Induced Tumours.

The rats used for this experiment were adult Canberra black male rats in which tumours had been induced by local irradiation of the flank skin with X rays from a Be window tube source (single skin dose was 4000*r*, HVD 1·0 to 2·0 millimetres in tissue) to a circular field 3·5 centimetres in diameter or by subcutaneous injection of 9,10-dimethyl 1, 2-benzanthracine in benzene solvent. The latent period for tumour growth was approximately six to eight months in each case, and animals were subjected to hydrogen peroxide therapy when tumours were well developed (one to two centimetres in diameter), fixed and growing progressively.

Hydrogen Peroxide.

Two brands of hydrogen peroxide were used. The first type was used in Experiment I (see Table I); in the case of the tumour-bearing rats, a solution of commercial hydrogen peroxide in water supplied by Townson and Mercer, Melbourne, was used, diluted to 0·5% (weight for volume) with distilled water. The second type was used in Experiments II, III and IV; it was a chemically pure solution of hydrogen peroxide supplied by Laporte Chemicals Proprietary Limited; a stock solution diluted to 30% (weight for volume) in water was used.

In conformity with the optimal concentrations recommended by Holman (1957), animals received a 0·5% solution (weight for volume) substituted for drinking water in the treated groups, except in Experiment III, in which the strength was doubled to 1·0% (weight for volume). The amounts of fluid consumed were measured daily for each cage of animals, except in Experiment I, in which the reduced consumption in peroxide-treated animals was first noticed (*vide infra*), and in the case of tumour-bearing rats. Each day the drinking bottles were emptied, washed and refilled with the solution of peroxide, which was stored under refrigerated conditions and was carefully measured for activity by standard permanganate titration from time to time.

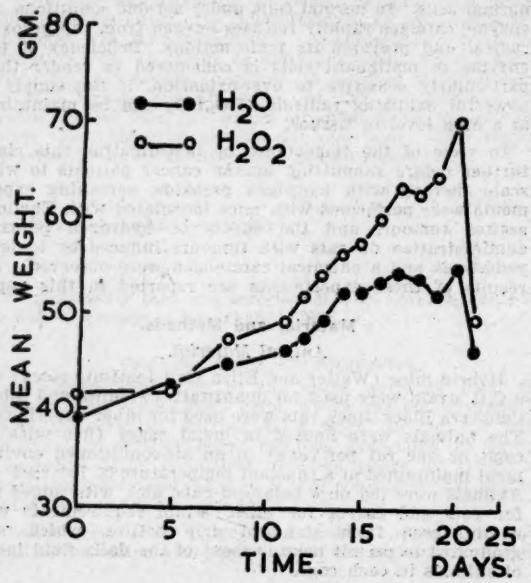

FIGURE I.

Mean animal weight changes in hybrid mice, after intraperitoneal inoculation with Ehrlich's ascites tumour (E.A.T.). Two groups of mice are shown treated with water and H_2O_2 (0·5% weight for volume) respectively (Experiment I).

Selection and Inoculation of Mice.

For each experiment (see Table I), litter mates were used and carefully selected, weighed and apportioned to the control and treated groups to minimize mean weight differences between groups in each experiment. The mean weight of the mice in each cage was estimated daily throughout the experiment and recorded (Figures II and III). Each mouse in Experiment I received an inoculation of 10,000,000 tumour cells taken from a single donor C$_3$H mouse seven days after passage with Ehrlich ascites tumour, and the mice in Experiments II, III and IV each received 1,650,000 cells from a similar single C$_3$H donor. The inoculation was intraperitoneal in Experiments I, II and III and subcutaneous in Experiment IV.

The inoculum size was measured by aspiration of the donor fluid; it was diluted in Tyrode solution at a temperature of 37·5° C., and the cell population was determined by counting in a Neubauer hæmocytometer.

Equal numbers of control and treated animals were used in each experiment. The details are set out in Table I.

Observations and Records.

Besides daily records of mean body weight and fluid intake in the mice, the deaths were recorded graphically in Experiments I to III, together with observable changes in animals. As each animal died, the abdomen was opened, the exudate was examined and dry smears were made which were then stained according to Leishman's technique and examined for tumour cells. A few selected exudates were examined by the aceto-orcein technique for chromosomal appearances. In Experiment IV, the development of the subcutaneous nodules was recorded, and the tumour size was measured at 14 days and later periods.

Rat Tumours.

Approximate estimations of tumour size before and after commencement of peroxide therapy were made in the six rats treated. In one animal which died 30 days after commencement of treatment, histological examinations of tissues were performed.

Results.

Ehrlich's Ascites Tumour.

The results of the various experiments and the statistical analysis are set out in Tables I and II and in Figures I to III. It will be seen that oral peroxide therapy had no significant effect on mortality from intraperitoneal inoculations of Ehrlich's ascites tumour (Table I). Also, peroxide therapy did not significantly alter the size of the tumour attained by subcutaneous inoculations of it, growing in its solid form.

The apparent beneficial effect of peroxide on the inoculated animals, suggested by a lessened gain in body weight, could be correlated with the decreased fluid intake in peroxide-treated animals and progressive dehydration (Figure II). The consumption of fluid by peroxide-treated animals in Experiment I was noticeably decreased, although not accurately measured from day to day. This result led to the measurement of fluid intake in the subsequent experiments. The mice used in Experiment I were a different strain and, more important, were older and twice the body weight in relation to mice used in Experiments II to IV. These older mice, despite restricted fluid intake, rapidly developed free fluid in the peritoneal cavity (Figure I), as distinct from the findings in younger animals. This finding was explained by the diminished powers of renal conservation of fluid in the young actively metabolizing mammal, and by an unavoidable water depletion, which resulted from a minimum excretory volume. This effect was also reflected in the weight changes recorded in Experiment IV in which the increase in body weight for control mice, which had been subcutaneously inoculated, paralleled that for non-inoculated mice of similar weight, whilst the peroxide-treated group showed a definite decrease in normal weight gain. The ratios (R) for increase in mean body weight for the paired groups in Experiments II to IV (Table II), compared with the ratio (R_1) for mean fluid consumption per unit of body weight, showed that a good correlation existed between the two ratios over the initial 14 day period, before the intraperitoneally inoculated animals became moribund. However, the closer approximation of R and R_1 for animals in Experiment IV ($R = 0·33$, $R_1 = 0·50$) was due to a falling off in fluid consumption by control animals intraperitoneally inoculated in Experiment II as tumour development progressed (Figures II and III).

In peroxide-treated, intraperitoneally inoculated mice, the amount of intraperitoneal fluid was greatly reduced; the fluid was much more viscous and "sticky" than that in water-treated animals, and a thick coating of tumour growth obscured the peritoneum and mesenteries. Examination of smears of this exudate revealed a dense population of tumour cells in each mouse, but no apparent differences in cellular morphology except close packing of the cells, which resembled the solid form of the tumour seen in subcutaneous implants. It is estimated that the amount of free fluid in the peritoneal cavity did not exceed one millilitre per mouse in the peroxide-treated groups in Experiments II and III, compared with four to five millilitres in the water-treated mice. In Experiment I (mice of higher initial body weight), the consistency, microscopic appearances and volume of the ascitic fluid were similar for both peroxide and water-treated animals.

Rat Sarcomata.

The soft-tissue sarcomata induced in rats by irradiation, or by 9, 10-dimethyl 1, 2-benzanthracine, failed to show any

TABLE Ia.

Experiment Number.[1]	Treatment.	Mouse Strain.[2]	Initial Mean Body Weight. (Grammes.)	Number of Animals.	Number of Deaths.	Mean Survival Time (±Standard Deviation) (Days.)	Significance.[3]	Mean Increase in Body Weight per Animal. (Grammes.)
I (Intraperitoneal inoculation.)	Controls (H_2O).	Walter and Eliza Hall hybrids.	40	10	10	19·3 (±2·7)	$t=1·3$	13
	Treated (H_2O_2, 0·5%).	Walter and Eliza Hall hybrids.	40	10	10	18·8 (±2·4)	$p>0·2$	14
II. (Intraperitoneal inoculation.)	Controls (H_2O).	C_3H	15	6	6	14·20 (±2·04)	$t=0·6$	12
	Treated (H_2O_2, 0·5%).	C_3H	15	6	6	15·00 (±2·10)	$p>0·5$	4
III. (Intraperitoneal inoculation.)	Controls (H_2O).	C_3H	15·5	6	6	16·30 (±2·33)	$t=0·06$	14
	Treated H_2O_2, 1·0%).	C_3H	15·5	6	6	16·20 (±2·48)	$p\simeq1·0$	2

TABLE Ib.

Experiment Number.[1]	Treatment.	Mouse Strain.	Initial Mean Body Weight. (Grammes.)	Number of Animals.	Number Developing Tumours.[4]	Mean Tumour Diameter at 20 Days. (Millimetres.)	Significance (t, p).	Mean Increase in Body Weight per Animal. (Grammes.)
IV. (Subcutaneous inoculation).	Controls (H_2O).	C_3H	14·7	6	5	12·64 (±1·56)	$t=1·81$	7·5
	Treated (H_2O_2, 0·5%).	C_3H	15·5	6	5	11·50 (±0·85)	$p>0·1$	2·5

[1] All animals in Experiment I received an intraperitoneal injection of 10,000,000 tumour cells, and animals in Experiments II and III, 1,650,000 tumour cells; animals in Experiment IV received a subcutaneous inoculation of 1,650,000 tumour cells.

[2] In each experiment, litter mates of the requisite weight were selected and apportioned to control and treated groups.

[3] The significance of the difference (p) in each experiment is calculated from Student's distribution t, applying the Bessel Correction $\left(\frac{n}{n-1}\right)$, and using the distribution

$$t=\frac{(\bar{X}-\bar{x})}{\sqrt{\left(\frac{1}{n_1}+\frac{1}{n_2}\right)\frac{n_1 s_1^2+n_2 s_2^2}{n_1+n_2-2}}}.$$

where $(\bar{X}-\bar{x})$ is the difference of the means, n_1, n_2 are the sample numbers, and s_1^2, s_2^2 are the respective sample variances.

[4] In one animal in each of the treated and control groups ascites resulted, apparently owing to an experimental error in entering the peritoneal cavity with the inoculating needle. Both animals died, at 18 and 19 days respectively, and are excluded from the analysis.

response to the peroxide treatment. All tumours grew progressively over a 30 day period of peroxide treatment to sizes four to five times the initial dimensions, with progressive infiltration and fixation of the neoplasm to muscle and other somatic structures. Death occurred during treatment in one animal and, at the present time (October, 1957) other animals are wasted and moribund. The animals consumed greatly diminished amounts of fluid (hydrogen peroxide) in comparison with the normal water intake.

Discussion.

Our results show that hydrogen peroxide administered orally to mice of two different strains inoculated with Ehrlich's ascites tumour (E.A.T.) fails to influence mortality from the tumour or to significantly retard synthesis of tumour tissue. In peroxide-treated mice which received subcutaneous inoculations of E.A.T. growing in a solid form, the tumours grew progressively at a rate not significantly different from that in water-treated animals. Sarcomata induced in rats by X irradiation or by a chemical carcinogen also failed to respond to oral hydrogen peroxide therapy. These results fail to parallel those of Holman (1957), in which the tumour screened was Walker 256 adenocarcinoma in rats. In view of this conflicting evidence, it is thought that further experimental studies should be undertaken before human patients who are suffering from malignant disease are submitted to treatment with hydrogen peroxide if palliative measures of reputable efficacy are still available.

An interesting finding which has resulted from our experiments is the marked reduction in free peritoneal fluid in young peroxide-treated mice. This effect is considered to be directly related to a decreased fluid intake and to the dehydration which results. It is accompanied by considerable general dehydration of tissues, which gives rise to retarded weight gain in growing animals.

The rationale on which peroxide treatment is based requires further critical examination from physiological and biochemical aspects. The instability of hydrogen peroxide in the presence of the enzyme catalase, which is present in considerable amounts in saliva and blood, raises problems in providing for an effective transfer of hydrogen peroxide from the oral cavity to the tumour cells, in order to establish adequate gradients and concentrations. Furthermore, the actual catalase content of various types of neoplasms needs to be estimated and correlated with that of normal cells in various organs.

Hydrogen peroxide-inactivating systems of the mammalian body include catalase, peroxidase and cytochrome-c peroxidase. These are transferring enzymes at all concentrations, and haem pigments also act as weak peroxidases. Catalase at low substrate concentrations also acts as a peroxidase, but at higher concentrations the reaction is represented by the decomposition of hydrogen peroxide to oxygen gas and water. Liver contains the most catalase, and it is decreased by tumour growth in distant sites. Red cells and kidney tissue also have high contents of this enzyme, but the presence of tumours has little effect on their enzyme constitution. The catalase content of tumours has not been investigated very thoroughly, but generally it appears to be low or non-existent.

In rats, the transplantable hepatoma 31, Jensen sarcoma and the transplantable epithelial tumour 2226 have virtually no catalase activity; in mice, with the exception

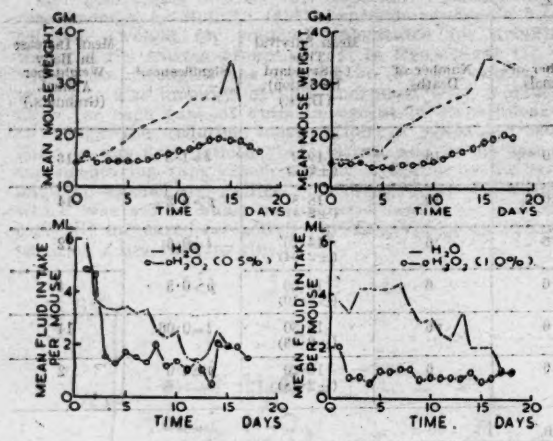

FIGURE II.

Mean weight changes and corresponding mean fluid intake in C3H strain mice after intraperitoneal inoculation with E.A.T. (Experiments II and III).

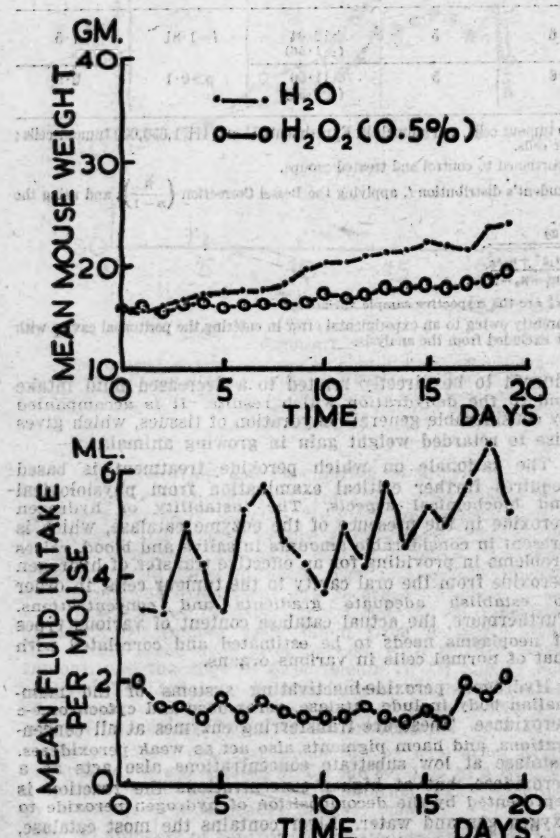

FIGURE III.

Mean weight changes and corresponding mean fluid intake in C3H strain mice subcutaneously inoculated with E.A.T. (Experiment IV).

of hepatomata, transplantable and spontaneously occurring tumours, which have been examined uniformly, show extremely low catalase activity (Greenstein, 1954).

Recently (Nickerson *et alii*, 1957), it has been shown that saliva contains both peroxidase and catalase, the former being a true secretion by the salivary glands, while the latter is presumed to be derived from oral bacteria. The chances that hydrogen peroxide will survive for any length of time in the body are rather remote.

TABLE II.

Experiment Number.	Ratio of Weight Gain in First 14 Days (G). $R=\frac{G\ (peroxide)}{G\ (control)}$.	Mean Daily Fluid per Gramme of Body Weight (*f*), Consumed Over First 14 Days. (Millilitres.)	Ratio of Mean Fluid Consumption $R_1=\frac{f\ (peroxide)}{f\ (control)}$.
I	$\frac{14}{13}$=1 (approx.)	—	—
II	$\frac{4}{12}$=0·33	0·2 / 0·1	0·50
III	$\frac{2}{14}$=0·14	0·2 / 0·06	0·30
IV	$\frac{2·5}{7·5}$=0·33	0·3 / 0·1	0·33

Conclusions.

1. Continuous oral administration of hydrogen peroxide to mice inoculated with Ehrlich's ascites tumour fails to influence mortality due to the tumour growing in the peritoneum, and it fails to influence the tumour size, attained by the solid form, in subcutaneously inoculated animals.

2. The reduction in weight of tumour-bearing animals treated with hydrogen peroxide is not due to inhibition of tumour growth, but is due to dehydration, which is also reflected by the decreased development of free peritoneal fluid, resulting in a "sticky", concentrated intraperitoneal exudate. This is seen particularly in young animals.

3. Despite the dehydration maintained in peroxide-treated animals over a 40 day period of continuous peroxide administration, mortality in the mice is not affected, although the normal increase in weight of treated mice is retarded.

4. The amounts of peroxide consumed over a 40 day period did not result in an identification of toxic symptoms in the mice.

5. The continuous administration of hydrogen peroxide to rats bearing soft tissue sarcomata, previously induced by either ionizing radiations or by 9,10-dimethyl 1,2-benzanthracine, failed to influence the development and growth rate of the tumours.

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Addendum.

In view of the fact that Holman's results were obtained with the use of commercial hydrogen peroxide in tumour-bearing rats, we examined the possibility that a preserva-

tive or contaminant in the commercial product was responsible for the inhibition of tumour growth. One such substance, commonly used to preserve the chemical stability of hydrogen peroxide, is methyl-*p*-hydroxy-benzoate. As we could find no account of this substance having been screened for anti-tumour activity, experiments were conducted with this substance and E.A.T. inoculated mice. As in previous experiments, the daily weight, the mean fluid intake and mortality were charted in both control and treated animals. Albino Swiss mice with an average body weight of 23 to 25 grammes were used, and they were inoculated intraperitoneally with 19,000,000 E.A.T. cells from a single donor C_3H mouse. The treated group were maintained on a solution of methyl-*p*-hydroxy-benzoate diluted to 0·25% (weight for volume) in water as drinking fluid. The results of this experiment are set out in Table III.

TABLE III.

Effect of Methyl-p-hydroxy Benzoate (MPHB) on Growth of Ehrlich Ascites Tumour in Albino Mice.

Group.	Mean Increase in Weight in First 14 Days. (Grammes.)	Mean Daily Fluid Intake per Animal in First 14 Days. (Millilitres.)	Survival Time (Days) (±Standard Deviation).
Controls	20·9	5·3	18·40±1·88
Treated with MPHB	10·7	5·8	18·00±3·23

The results show that, whilst methyl-*p*-hydroxy-benzoate in the concentrations administered orally failed to influence the mortality in this experiment, the gain in weight of treated animals was only half that of controls, despite an essentially comparable fluid intake. The explanation for the latter is uncertain, and further experiments are in hand which are designed to determine whether this is due to a diuretic action of methyl-*p*-hydroxy-benzoate or to an initial effect on establishment of the tumour inoculum.

CHRONIC PULMONARY TUBERCULOSIS IN KOREA: SUMMARY OF 164 CASES.

By J. N. BURGESS, D.D.R.,
Korea Church World Service, Seoul, Korea.

IN January, 1954, a chest clinic was opened at the Severance Hospital, Seoul, under the auspices of the Korea Church World Service. This was the first chest clinic for the out-patient treatment of pulmonary tuberculosis established in South Korea. Since then the Korea Church World Service has organized 12 other clinics, and the Korean Government has opened about 130 similar clinics. After routine examinations, the taking of X-ray films of the chest and sputum tests, those diagnosed as tuberculous are given appropriate drugs. They are then interviewed by a public health nurse, who gives instructions about the danger of infecting others, and the need for complete bed rest and isolation is stressed, though this is often impracticable. In a series of 1000 cases in Seoul, it was found that 650 of the patients were each living with family units of up to five people in one room, and that 220 were living in two rooms measuring six by six or eight by 10 feet. Thus overcrowding increased the risk of infection, and under-nourishment and insufficient rest retarded the patient's recovery. The nurses' home-visiting team called at each new patient's house and investigated the conditions. Several families were able to build a small room for the patient to live in. The nurses also visit the homes at regular intervals to help and advise the patient and his family, who are urged to be radiologically examined as soon as possible.

These clinics treat about 50,000 patients, who represent only about 10% of the estimated number of cases of pulmonary tuberculosis in the country. Only 4000 sanatorium beds are available, and many of these are occupied for an indefinitely long period by people with chronic tuberculosis. Korea Church World Service clinics are treating 5000 out-patients and have 200 sanatorium beds; in these most patients are kept for only six months, and patients with early and active disease are given preference.

The death rate among tuberculous out-patients is 2% *per annum*, and in the sanatoria it is 5%, while the estimated tuberculosis death rate among tuberculous patients for the whole of Korea is 10%, or 300 per 100,000 population, with a total of 60,000 deaths per year.

Since 1951 tuberculin testing has been carried out on a large scale; 3,200,000 persons have been tested, and 1,625,000 have received B.C.G. inoculations. The positive reaction rates rise from 10% at the age of one year to 80% at the age of 18 years. The average rate among children is 50%.

In the out-patient clinics, 21% of the patients had minimal disease, 40% moderately advanced disease and 39% far advanced disease. In an X-ray survey of 470,000 students, 2% showed radiological evidence of active disease; of these, 75% had minimal disease, 21% moderately advanced disease and 4% far advanced disease. Among 310 contacts, 18% were found to have active tuberculosis.

Recently a survey was made of the records of 164 patients who had started treatment at Severance Hospital Chest Clinic in 1954 or 1955, and thus had received at least two years' medical treatment. The majority did not continue; 50% dropped out in the first three months, as they had come to the clinic for diagnosis only, and went elsewhere for treatment. Another 40% ceased coming for reasons which could not be determined, only a few being discharged as cured. Of the 164 patients, 80 (50%) had far advanced disease, 68 (40%) had moderately advanced disease and 16 (10%) had minimal disease. The sputum tests showed 116 to be positive reactors when they first came to the clinic, and of this number 59 are still positive reactors. Of the other 57 (50%), eight (7%) became "negative" in three months, 16 (14%) in six months, 11 (9%) in 12 months, nine (8%) in 18 months and 13 (12%) in 24 months. Four changed from "negative" to "positive".

The drugs used were isoniazid and PAS for 72 patients, isoniazid and streptomycin for six, and isoniazid and PAS, later changed to streptomycin and PAS, for 66. Of those still "positive", only 10 had not had the three drugs.

The X-ray changes were checked every three months, with the following results: 60 patients (36%) showed no change, 39 (25%) showed marked improvement, 56 (34%) showed moderate improvement, and nine (5%) showed deterioration. Thus 59% showed radiological improvement under treatment. The final X-ray films were scanned, and the resultant condition was assessed as follows: 96 (57%) had residual fibrosis, 34 (20%) had atelectasis, 17 (10%) had cavities, five (3%) had spreading infiltration and 12 (8%) had clear lungs.

The classification of fibrosis varied from "minimal" to "extensive bilateral involvement". Atelectasis referred to collapse of the upper lobe, and a few patients had apparent atelectasis of the whole lung. Lateral films and tomograms were not available, so the figures for atelectasis and cavities are probably low. From these latest films an estimate of possible surgical intervention was made, provided that the lesions were mainly unilateral, and 30 (18%) of patients were considered suitable for surgery.

This series of 164 cases of chronic pulmonary tuberculosis gives some indication of the problem of chronicity. Of the patients with moderate and advanced disease, 50% still have tubercle bacilli in their sputum after two years of medical treatment with the three drugs used, 36% showed no radiological improvement, and another 5% showed deterioration. Although 18% could be cured by surgery, it is not likely that more than 1% will be able to be operated on, owing to inadequate surgical facilities and the patients' inability to pay even nominal fees.

Discussion.

The problem is how to improve the recovery rate in this country. Undoubtedly tuberculin testing and B.C.G. inoculations are valuable, and these should be used more, especially for new-born babies and pre-school children.

Mass radiography would discover many people with early, asymptomatic lesions, who could be completely cured. But what about those with moderately advanced and far advanced lesions? Should larger doses of isoniazid be given with daily injections of streptomycin? Or are some less toxic, newer drugs going to give better results? The need for absolute bed rest and for a nutritious diet, instead of simply rice and soup, should be stressed. Increased surgical facilities must be available, and should be free of charge to those who cannot afford to pay.

Of the 164 patients, there are still 120 with chronic disease. Can isoniazid keep their disease from becoming reactivated? If the sputum continues to contain bacilli, are the organisms so attenuated that they cannot cause active disease in contacts? We are reminded by this series that pulmonary tuberculosis is still a chronic disease, which can be cured clinically, radiologically and bacteriologically in only a small percentage of cases when it is moderately advanced or far advanced.

Summary.

1. Chest clinics have been established in South Korea since January, 1954, to treat pulmonary tuberculosis on an out-patient basis.

2. Treatment is being given to 50,000 patients in 13 Korea Church World Service Clinics and 130 government clinics, and there are 4000 in sanatoria.

3. Nurses visit the homes regularly and advise on isolation, prevention of infection and diet.

4. Overcrowding is prevalent; for example, of 1000 patients, 650 were each living in one room measuring six by six or eight by 10 feet with family units of up to five persons.

5. The death rate among tuberculous patients in the out-patient clinics is 2%, in the sanatoria 5% and in the general population 10%; this amounts to 300 per 100,000, or 60,000 deaths per year in the whole of Korea.

6. Tuberculin tests produce 50% of positive results in children, and up to 80% at the age of 18 years. B.C.G. vaccination has been given to 1,625,000 children.

7. An X-ray survey of 470,000 students showed that 2% had active tuberculosis; of these, 75% had minimal lesions, 21% moderately active disease and 4% far advanced disease. Of 310 contacts, 18% had active disease.

8. A total of 164 chronic cases is analysed. Of these patients, 50% had far advanced disease, 40% moderately advanced disease and 10% minimal disease.

9. After two years' treatment with three drugs, 50% of patients still had tubercle bacilli in their sputum.

10. No X-ray change was found in 36% of patients, and in 5% deterioration had occurred.

11. Residual fibrosis was found in 57%, atelectasis in 20%, cavitation in 10%, increasing infiltration in 5% and normal appearances in 8%.

12. Of these patients, 18% could be cured by operation if facilities and funds were available. The others have chronic, potentially active disease, and are possibly infectious.

Reviews.

The Glaucomas. By H. Saul Sugar, M.D., F.A.C.S.; Second Edition; 1957. New York: Paul B. Hoeber. 9¼" × 6", pp. 528, with 164 illustrations. Price: $13.50.

The second edition of "The Glaucomas" by Sugar has grown from a small book, which originated as a series of lectures to post-graduate students in ophthalmology, to a treatise on glaucoma. In the six years since the first edition, many notable advances have been made in the understanding and treatment of glaucoma. Sugar has rewritten the book incorporating these new advances, and the new additions to the text are supported by an increase in the number of references to the literature which appear at the end of each chapter.

Tonography is discussed in relation to its value in the diagnosis of early glaucoma and in the evaluation of treatment in the proven cases. We cannot but feel that this procedure is now passing out of the purely research into the clinical field, and it should be available in the diagnosis of the doubtful case.

The use of "Diamox" is described. Sugar is careful to indicate that the drug is not considered best for long-term therapy—a contention which will have the support of most ophthalmologists. This is especially so in narrow-angle glaucoma, in which it should be used only in preparation for surgery, for here its use may lower tension while peripheral anterior synechiæ continue to form.

Although he mentions the use of peripheral iridectomy in the fellow eye of a patient who has suffered an attack of acute narrow-angle glaucoma, Sugar has not given it the emphasis which would convince the hesitant that this procedure is the best safeguard against an attack in that eye. In the surgical treatment of narrow-angle glaucoma, we should like to see more emphasis placed on the technique of peripheral iridectomy which has superseded the basal iridectomy.

In spite of these minor criticisms, Sugar's book will probably become the standard text-book on glaucoma, and should be read by all practising ophthalmologists.

Gastro-Intestinal Obstruction. By Meyer O. Cantor, M.D., M.S., F.A.C.S., and Roland P. Reynolds, M.D., F.A.C.S.; 1957. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 10¼" × 7¼", pp. 580, with 415 illustrations. Price: £9 18s.

In this book there can be no question that nothing is left out, from *dysphagia lusoria* and œsophageal atresia at one end, to lymphogranuloma and a wayward "pop" bottle at the other. There is, in between, a formidable list of possible causes of intestinal obstruction, and even the rarest is given more than passing attention. Obstruction in the young, obstruction in the aged, obstruction in the pregnant, obstruction after gastrectomy—it is all here. The result is an encyclopædic work, and to do justice to this review we were committed to a great deal of very tedious reading.

It is inevitable that, with Cantor as the senior author, a great deal of attention should be paid to the technique of gastro-intestinal suction, and here again the coverage is complete, with a description of every form of tube, short or long, introduced since the turn of the century.

The volume is lavishly produced on thick glossy paper and the text is profusely illustrated; but many of the X-ray photographs, which abound, are badly reproduced and by no means immediately informative. For any surgeon who can afford to add to his shelves a dull book of reference, it will serve very well; but if he wants rather a concise and practical guide to the management of intestinal obstruction, he should certainly look elsewhere.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Hypnography: A Study in the Therapeutic Use of Hypnotic Painting", by Ainslie Meares, M.B., B.S., B.Agr.Sc., D.P.M.; 1957. Springfield, Illinois, U.S.A.: Charles C. Thomas. Oxford: Blackwell Scientific Publications. 9" × 5¾", pp. 288, with many illustrations. Price: 58s. 6d. (English).

The author, a Melbourne psychiatrist, describes this as essentially a clinical study.

"Recent Advances in Oto-laryngology", Third Edition, by F. Boyes Korkis, M.B., Ch.B., D.L.O., F.R.C.S., F.R.C.S. (Eng.), with a foreword by R. Scott Stevens, M.D., F.R.C.S.; 1958. London: J. and A. Churchill, Limited. 8¼" × 5", pp. 446, with two coloured plates and 144 text figures. Price: 60s. (English).

The previous edition was published in 1949. The present edition has a new author.

The Medical Journal of Australia

SATURDAY, MAY 10, 1958.

ONE PROFESSION.

THE President of the New South Wales Branch of the British Medical Association, Dr. A. W. Morrow, has raised vital issues in his presidential address published in this issue (see page 621). Being also a Fellow of a senior Royal Australasian College he is in a position to see both sides of the question of relationships within the medical profession, and it is not to be expected that his quiet but frank comments will please everyone. However, he has said what needed to be said and has said it well.

His first contribution is to put in its right perspective the relationship between the British Medical Association and the Colleges in Britain and in Australia. There has been a good deal of sympathetic feeling in Australia over certain medico-political happenings in England involving the Association and the Colleges, and in our view much of this feeling is thoroughly justified. At the same time there are sound reasons why many members of the profession in Britain prefer to look to the Colleges, especially the senior Royal Colleges, for their leadership. The situation in Australia is, as Dr. Morrow states, essentially different, and it would be neither wise nor logical for the Association in Australia and the Australasian Colleges to allow their attitude to one another to be influenced by either recent events in Britain or the relative status of the several medical bodies there. In Australia the Association by seniority, by constitution and by experience is best fitted to deal with medico-political matters, especially negotiations with governments, and we hope that this will come to be universally acknowledged. There is a place in its councils for members of all sections of the profession who by their wisdom and experience gain the confidence of their colleagues. It is in no sense a sectional body and ceases to be fully representative of the profession only by the default and secession of individuals or groups. The Federal Council has tried hard to find a formula that will safeguard the interests and satisfy the aspirations of all sections of the profession and will at the same time ensure efficient machinery for medico-political negotiations, and it is gratifying to note that real progress has been made in the discussions with the Colleges and other special groups on this matter. There is indeed nothing lacking in the present formula that cannot be covered by mutual goodwill and common sense.

Dr. Morrow has also made important references to the scientific activities of the Association in relation to other bodies within the profession. It is to be hoped that this will provoke vigorous and constructive discussion. The scientific side of the Association's programme has always been prominent and must remain so for many good reasons. It would be a retrograde, even a dangerous step for the Association to become just a medico-political body; and especially in dealing with outside bodies and people who seek guidance in medical matters, no special section of the profession could very well take its place. However, there is room for some hard thinking and for an honest attempt to dispense with or modify anything that serves no useful purpose. Many people feel, for example, that the cities at least are saturated with medical meetings, and that something should be done to prune the rambling vine in the interests of better fruit. A frank exchange of views on this question among the six Branches of the Association, with a pooling of their experience, might well be illuminating and could provide a useful background for relevant discussions with sectional groups, post-graduate committees and others. By all means let the Association see that its members are catered for scientifically, but let it explore the possibility that this may sometimes at least be done best by negotiation with other bodies which have already done much to raise the standard of medicine in Australia. The library services of the Association are, as Dr. Morrow points out, most important and should be developed as fully as possible. Other bodies with limited resources might well be induced to relinquish to the Association their activities in this field, except perhaps for certain specialized collections. The Association's triennial congresses are also of great importance. Their role and particularly their detailed organization have been under review for some time, and the feeling has been growing that the keynote should be more combined and less sectional activity. It was mainly for this reason that, with the utmost goodwill, it was decided not to accept the suggestion of the College of General Practitioners that a Section of General Practice be added to the list of sections. The great value of the congress is the opportunity it offers for an exchange of ideas among all sections of the profession, and the combined meeting can be just as acceptable to the specialist who wants to keep informed of matters outside his own specialty as it is to the general practitioner. By the same token the inclusion of an experienced general practitioner in the panel of speakers at a combined meeting can be to the advantage of everyone and might be considered more often.

This journal can, we hope, play a useful part in this situation. It is perhaps uniquely fitted to do so. The child of the Association, it is firmly loyal to its parent; yet, because of the way in which it is constituted, it is not bound by parental apron strings. By the grace of the Association, it is free to serve the whole profession. This it wishes to do in any way that it can, and it invites all sections of the profession to accept that this is so. In particular, its correspondence columns are an open forum for the full and frank discussion of such matters as we have been considering.

Long ago among the Greeks, to whom our profession owes so much, there was a fable in the tradition of Æsop about a farmer who had two quarrelsome sons. Nothing he could say seemed able to stop them fighting one another. Then he handed them a bundle of sticks and asked them to break it. Neither of them could do so. When, however, he handed them the sticks one at a time,

they broke them with ease, and the lesson was learnt. Divided they were weak, together they were strong. Medicine today is being challenged in many parts of the world. The threat is not only on the medico-political level, involving the interests of the medical profession; it also involves the standards of medical practice and the duty that the good doctor knows that he owes to his patient. A divided profession is likely to succumb to this threat. A united profession need have no such fear.

Current Comment.

MALARIA: THE WORLD'S MOST EXPENSIVE DISEASE.

A SURVEY of ninety-nine countries and territories has just been completed by the World Health Organization as a first step in the most ambitious war on disease ever attempted. The disease is malaria. The aim is its complete annihilation from the face of the earth. Already, according to a recent WHO communication, eradication has nearly been achieved in nine countries with a population of 231,000,000, and is well advanced in large areas of seven others (43,000,000 population). In still another 44 countries (302,000,000 population) a beginning has been made, and in 16 with a population of 580,000,000 eradication is being planned.

When WHO was set up in 1948 malaria was given top priority in its programme. Faith was placed mainly in campaigns of house spraying with DDT. Then the first warning appeared that all was not well. In 1951, one type of malaria mosquito stopped dying when exposed to DDT: then another, then still another. Resistance had set in. Most of the countries concerned had planned on maintaining control indefinitely with DDT spraying. Another approach was needed, and in 1955 enough evidence had been accumulated to point to a new policy: total eradication—that is, definite elimination of the disease by an extensive campaign limited in time. The essence of the plan is to break the cycle of transmission for long enough to allow the parasite to die out.

According to WHO, eradication has been accomplished and is standing the test of time in several countries—U.S.A., Puerto Rico, Chile, Cyprus, Corsica and Italy. In other areas, eradication campaigns are on the verge of success. In southern Europe, 4,000,000 new cases of malaria a year have become less than 10,000. In many countries, the way ahead to eradication can be seen clearly. Malaria deaths, 3,000,000 in 1946, declined to 2,000,000 in 1955, and perhaps 1,000,000 in 1957.

Altogether, 51,000,000 people in nine countries have been freed from the scourge of malaria. By 1965, it is possible that 550,000,000 at present at risk will be free. For the next five years, WHO will need $32,000,000 to give necessary help to countries undertaking malaria eradication. Pledges amounting to $17,000,000 have been received. The shortage is $15,000,000. WHO is empowered to ask funds not only from governments, but from individuals, and from foundations, from industry and from labour organizations. The United States Government has already primed the pump with a contribution of $5,000,000 and expects to contribute still more.

WHO is convinced that world malaria eradication is not only feasible: it is essential. In fact, since mere control cannot be relied upon indefinitely, eradication is the only practical way of tackling malaria. It is also the only economic way. The benefits are enormous. In Mexico today, the loss of working time due to malaria is estimated at $18,000,000 a year. The total cost of malaria eradication in that country within the next five years will be $21,000,000. In India, loss of wage-earning capacity due to customary six-day attacks of malaria is estimated at $30,000,000. Since malaria decreases efficiency by about 25% during the whole year, loss on this account increases the total loss to $500,000,000 a year. The total cost of malaria eradication in India by 1965 has been estimated at $114,000,000. In the Philippines, malaria control has rendered possible road construction, mining, logging and other industrial projects as well as agricultural settlement in formerly stricken areas. The effect, though not estimated in money terms, has been to transform the economy in several islands. Malaria is also a cause of economic loss to countries which import goods from malarious areas. Many of the world's important raw materials—basic minerals, hardwood, coffee, cocoa, fruit etc.—come from countries where the cost of production is inflated by the inefficiency and invalidism of the population. Dr. Paul F. Russell, of the Rockefeller Foundation, has estimated that this amounts to a "hidden tax" of at least 5% paid by the importing countries. WHO believes that capital invested in malaria eradication will be regained by the community in a few years, perhaps within a year. This capital can then be used for the general development programme which follows freedom from malaria. Development funds find their way back to industry. Improvement of health standards also implies increased demand for consumer goods. In other words, "malaria eradication is also good business". More important is the fact that infant mortality decreases with malaria eradication, and that human well-being increases. The value of that is inestimable.

OBESITY—WHY?

EVERYONE will agree that food, obesity, over-eating and calories are inextricably related; but why do so many people over-eat and why do so many become obese? Why do many others who seem to eat too much remain quite lean? A panel of seven experts—a physician, a professor of nutrition, a physiologist, a clinical professor of medicine, a lecturer in psychoanalysis, an associate professor of psychiatry and an assistant in psychiatry—discuss these problems, each from his own point of view, in the January number of *International Record of Medicine*.[1] The physiologist, Jean Mayer, discusses the experimental aspects of obesity. He points out that appetite is controlled indirectly through the ventro-medial nuclei of the hypothalamus, and when these are destroyed in animals there is an elimination of satiety feelings, and the animal does not stop eating. In animals with the obese hyperglycæmic syndrome there is hypersecretion of glucagon and insulin, with a big increase in the liver glycogen turnover and increased fat production. Experimental obesity in animals is of two main types, the regulatory type, seen only when over-eating takes place, and the metabolic type, in which lipogenesis can occur even in the fasting state. M. G. Goldner discusses the relation between obesity and diseases. He points out that obesity does not cause diseases like diabetes, coronary disease and Cushing's disease, but may aggravate them. N. Jolliffe states: "Diet is the only treatment for obesity, but this treatment is not given in a vacuum. Obtaining and developing a caloric deficit is the only possible therapy; every other method is only an adjunct." E. Bergler discusses the psychological reasons for over-eating, but without reaching any firm conclusions. He states that all obese patients are "injustice collectors". H. Bruch very briefly discusses the psychiatric aspects of obese patients who are not "motivated to lose weight". He points out that obesity may be associated with a variety of psychiatric conditions, and it is not possible to group together the different types. In many persons being fat is an alternative to a psychosis. A. Wolf prefers to enlist the obese patient's ego before commencing dietetic treatment, and he claims that it is necessary to use psychotherapy to strengthen the ego. It seems to be generally agreed that many patients are happier when they are fat and should be left alone.

[1] *Int. Rec. Med.*, 1958, 171: 1 (January).

As an addendum, Jean Meyer presents a paper entitled "A Physiologist Examines Some Psychiatric Assumptions Concerning Obesity". He states that the basic thermodynamic fact in obesity is that for any substantial deposition of fat to take place more energy has to be consumed than is expended. Most workers concerned with the psychiatric or the psychosomatic aspects of obesity have assumed that this means that the obese patient eats quantities of food much in excess of those eaten by persons of normal weight. They have therefore tried to account for the hyperphagia. Obese children and adolescents commonly eat less than non-obese subjects, but they also expend far less energy in play activities. The same applies to very many adult obese subjects, and inactivity may well be the most frequent cause of "creeping" over-weight in highly urbanized society. This does not mean that some obese persons do not eat amounts of food far in excess of normal, but very large eaters seem to be the exception. The causes of the inactivity are still unknown. It had been assumed that appetite and the regulation of food intake were purely psychological phenomena, and that psychological investigation alone was needed to elucidate the problem of obesity. This is now known to be far from true. Much work has been and is being done on the role of various hypothalamic centres in regulating fat production and storage. A single injection of gold-thioglucose in animals will produce obesity due to selective destruction of cells in the ventro-medial area of the hypothalamus. There are no doubt other regulatory mechanisms. There are forms of obesity which are purely psychogenic, but they have to be distinguished more clearly from those that are not.

THE TREATMENT OF INTRACTABLE PAIN.

THE treatment of intractable pain in certain diseases and in the terminal stages of others almost always presents problems. J. S. Richardson,[1] in a symposium on the treatment of pain at the South African Medical Congress in 1957, presented a paper in which he reviewed the use of a number of the newer drugs in the treatment of intractable pain.

The adrenal steroid hormones are very useful for the pain in acute rheumatoid arthritis, provided that the difficulties associated with their use are always taken into account. The pain of acute gout which has failed to respond to the more usual measures will often react dramatically to cortisone, although there may be relapse when the cortisone is withdrawn; but this may be prevented by colchicine. Steroid hormones and corticotropin, in combination with alkylating agents such as triethylene melamine and nitrogen mustard, will often control intense pain where there are widespread metastases, and in Hodgkin's disease and follicular lymphoma. Adequate hæmatological control is important in these cases. Anti-metabolite drugs such as 6-mercaptopurine, which has practically no toxicity, are useful in acute leukæmia, with or without cortisone or corticotropin. The sex hormones are useful in some forms of cancer. Destruction of the pituitary by implantation of radioactive gold or yttrium is being tried in several centres in cases of advanced cancer.

Deep X irradiation is of great value in controlling pain in some metastatic deposits in bone. Irradiation of the parotid gland in the early stages of acute parotitis is effective in controlling pain. This is particularly useful when the patient is in a severely debilitated state. Whatever other methods are used, sooner or later the use of analgesic drugs will play some part in the treatment of chronic pain. The value of mild analgesics is sometimes greater than is commonly realized. Richardson quotes H. K. Beecher, who showed that "10 gr. of acetylsalicylic acid was demonstrably more effective as an analgesic than 1/6 gr. of morphia or 1 gr. of codeine, all by mouth". The relief of anxiety is a first step in the management of chronic pain. To this end chlorpromazine has been used for some time, but large doses are required. About 150 milligrammes in 24 hours are required in doses of 25 milligrammes taken by mouth four times a day with 50 milligrammes at night. While chlorpromazine has itself no analgesic effect, its use lessens the quantity of analgesic required, besides improving the mental attitude of the patient.

Morphine or one of its substitutes will be required in many cases. Methorphinan ("Dromoran") is particularly useful, as it can be given effectively by mouth, and it causes less nausea than morphine. The respiratory depression which is produced by morphine, pethidine and "Dromoran" can be reduced by levallorphan tartrate in doses of approximately 0·5 to 1·5 milligrammes. For increasing the effectiveness of morphine and diminishing its ill effects in the control of intractable pain in the terminal stages, amiphenazole ("Daptazol") is useful, particularly when large doses of morphine (1·0 to 2·5 grains) are being used. Respiratory depression, narcosis and reduction in the cough reflex are said not to occur. While morphine or its substitutes will often have to be given towards the end, it is not always necessary to resort to injections immediately and thus abandon the great advantages of oral medication. The object, Richardson states, is to prevent the agonizing pain, but not necessarily to abolish pain entirely (which may result in severe clouding of consciousness alternating with periods of agony), so that our patients may die with all possible dignity as well as in peace.

[1] *South African M. J.*, 1958, 32:158 (February 8).

ARTIFICIAL INSEMINATION IN THE HUMAN.

THE urge to parenthood is one of the strongest drives and its denial one of the greatest deprivations. In Genesis, Rachel was barren and said to Jacob: "Give me children, or else I die." This feeling is echoed in the hearts of many women, and when the male partner is at fault the anguish is intensified for both. This private state of affairs is only too obvious to those interested in the subject, and to the physician, dedicated to the relief of physical and psychic illness and wishing to contribute to the sum of total human happiness, heterogenous insemination carried out with certain safeguards might seem the ready answer. Such a private solution, while it may satisfy, has obligations of a moral and legal nature extending to the rest of society, and involving the donor, the unborn child and the doctor. Since the matter lies in the field of sex and is emotionally charged, attitudes are as varied as religious creeds.

In his book, "Artificial Insemination in the Human",[1] Dr. A. M. C. M. Schellen has produced a work on the subject which has interests extending beyond the specialist, especially as in the U.S.A. the procedure has been performed 100,000 times. Australia is mentioned as one of the countries in which it is finding increasing use, but one may wonder on what evidence this statement is based. The book is unusually comprehensive with exhaustive references, and is a full and frank study of the history and medical techniques, as well as the religious, ethical and sociological problems involved, the last-mentioned aspects still being in the formative stage.

From many points of view, the subject is a controversial one, and one of the merits of Schellen's book is that he, careful not to decide the issues, is able to remain detached and objective. After 400 pages of impartial and documentary research, it comes almost as a revelation, in the concluding epilogue, to know that Dr. Schellen's personal attitude, on the weight of evidence and moral grounds, is averse to the procedure.

[1] "Artificial Insemination in the Human", by A. M. C. M. Schellen, M.D., with an Introduction by Sophia J. Kleegman, M.D.; 1957. Amsterdam, Houston, London and New York: Elsevier Publishing Company. 9" × 6", pp. 438, with 10 illustrations. Price: 72s. (English).

they broke them with ease, and the lesson was learnt. Divided they were weak, together they were strong. Medicine today is being challenged in many parts of the world. The threat is not only on the medico-political level, involving the interests of the medical profession; it also involves the standards of medical practice and the duty that the good doctor knows that he owes to his patient. A divided profession is likely to succumb to this threat. A united profession need have no such fear.

Current Comment.

MALARIA: THE WORLD'S MOST EXPENSIVE DISEASE.

A SURVEY of ninety-nine countries and territories has just been completed by the World Health Organization as a first step in the most ambitious war on disease ever attempted. The disease is malaria. The aim is its complete annihilation from the face of the earth. Already, according to a recent WHO communication, eradication has nearly been achieved in nine countries with a population of 231,000,000, and is well advanced in large areas of seven others (43,000,000 population). In still another 44 countries (302,000,000 population) a beginning has been made, and in 16 with a population of 580,000,000 eradication is being planned.

When WHO was set up in 1948 malaria was given top priority in its programme. Faith was placed mainly in campaigns of house spraying with DDT. Then the first warning appeared that all was not well. In 1951, one type of malaria mosquito stopped dying when exposed to DDT: then another, then still another. Resistance had set in. Most of the countries concerned had planned on maintaining control indefinitely with DDT spraying. Another approach was needed, and in 1955 enough evidence had been accumulated to point to a new policy: total eradication—that is, definite elimination of the disease by an extensive campaign limited in time. The essence of the plan is to break the cycle of transmission for long enough to allow the parasite to die out.

According to WHO, eradication has been accomplished and is standing the test of time in several countries—U.S.A., Puerto Rico, Chile, Cyprus, Corsica and Italy. In other areas, eradication campaigns are on the verge of success. In southern Europe, 4,000,000 new cases of malaria a year have become less than 10,000. In many countries, the way ahead to eradication can be seen clearly. Malaria deaths, 3,000,000 in 1946, declined to 2,000,000 in 1955, and perhaps 1,000,000 in 1957.

Altogether, 51,000,000 people in nine countries have been freed from the scourge of malaria. By 1965, it is possible that 550,000,000 at present at risk will be free. For the next five years, WHO will need $32,000,000 to give necessary help to countries undertaking malaria eradication. Pledges amounting to $17,000,000 have been received. The shortage is $15,000,000. WHO is empowered to ask funds not only from governments, but from individuals, and from foundations, from industry and from labour organizations. The United States Government has already primed the pump with a contribution of $5,000,000 and expects to contribute still more.

WHO is convinced that world malaria eradication is not only feasible: it is essential. In fact, since mere control cannot be relied upon indefinitely, eradication is the only practical way of tackling malaria. It is also the only economic way. The benefits are enormous. In Mexico today, the loss of working time due to malaria is estimated at $18,000,000 a year. The total cost of malaria eradication in that country within the next five years will be $21,000,000. In India, loss of wage-earning capacity due to customary six-day attacks of malaria is estimated at $30,000,000. Since malaria decreases efficiency by about 25% during the whole year, loss on this account increases the total loss to $500,000,000 a year. The total cost of malaria eradication in India by 1965 has been estimated at $114,000,000. In the Philippines, malaria control has rendered possible road construction, mining, logging and other industrial projects as well as agricultural settlement in formerly stricken areas. The effect, though not estimated in money terms, has been to transform the economy in several islands. Malaria is also a cause of economic loss to countries which import goods from malarious areas. Many of the world's important raw materials—basic minerals, hardwood, coffee, cocoa, fruit etc.—come from countries where the cost of production is inflated by the inefficiency and invalidism of the population. Dr. Paul F. Russell, of the Rockefeller Foundation, has estimated that this amounts to a "hidden tax" of at least 5% paid by the importing countries. WHO believes that capital invested in malaria eradication will be regained by the community in a few years, perhaps within a year. This capital can then be used for the general development programme which follows freedom from malaria. Development funds find their way back to industry. Improvement of health standards also implies increased demand for consumer goods. In other words, "malaria eradication is also good business". More important is the fact that infant mortality decreases with malaria eradication, and that human well-being increases. The value of that is inestimable.

OBESITY—WHY?

EVERYONE will agree that food, obesity, over-eating and calories are inextricably related; but why do so many people over-eat and why do so many become obese? Why do many others who seem to eat too much remain quite lean? A panel of seven experts—a physician, a professor of nutrition, a physiologist, a clinical professor of medicine, a lecturer in psychoanalysis, an associate professor of psychiatry and an assistant in psychiatry—discuss these problems, each from his own point of view, in the January number of *International Record of Medicine*.[1] The physiologist, Jean Mayer, discusses the experimental aspects of obesity. He points out that appetite is controlled indirectly through the ventro-medial nuclei of the hypothalamus, and when these are destroyed in animals there is an elimination of satiety feelings, and the animal does not stop eating. In animals with the obese hyperglycæmic syndrome there is hypersecretion of glucagon and insulin, with a big increase in the liver glycogen turnover and increased fat production. Experimental obesity in animals is of two main types, the regulatory type, seen only when over-eating takes place, and the metabolic type, in which lipogenesis can occur even in the fasting state. M. G. Goldner discusses the relation between obesity and diseases. He points out that obesity does not cause diseases like diabetes, coronary disease and Cushing's disease, but may aggravate them. N. Jolliffe states: "Diet is the only treatment for obesity, but this treatment is not given in a vacuum. Obtaining and developing a caloric deficit is the only possible therapy; every other method is only an adjunct." E. Bergler discusses the psychological reasons for over-eating, but without reaching any firm conclusions. He states that all obese patients are "injustice collectors". H. Bruch very briefly discusses the psychiatric aspects of obese patients who are not "motivated to lose weight". He points out that obesity may be associated with a variety of psychiatric conditions, and it is not possible to group together the different types. In many persons being fat is an alternative to a psychosis. A. Wolf prefers to enlist the obese patient's ego before commencing dietetic treatment, and he claims that it is necessary to use psychotherapy to strengthen the ego. It seems to be generally agreed that many patients are happier when they are fat and should be left alone.

[1] *Int. Rec. Med.*, 1958, 171: 1 (January).

As an addendum, Jean Meyer presents a paper entitled "A Physiologist Examines Some Psychiatric Assumptions Concerning Obesity". He states that the basic thermodynamic fact in obesity is that for any substantial deposition of fat to take place more energy has to be consumed than is expended. Most workers concerned with the psychiatric or the psychosomatic aspects of obesity have assumed that this means that the obese patient eats quantities of food much in excess of those eaten by persons of normal weight. They have therefore tried to account for the hyperphagia. Obese children and adolescents commonly eat less than non-obese subjects, but they also expend far less energy in play activities. The same applies to very many adult obese subjects, and inactivity may well be the most frequent cause of "creeping" over-weight in highly urbanized society. This does not mean that some obese persons do not eat amounts of food far in excess of normal, but very large eaters seem to be the exception. The causes of the inactivity are still unknown. It had been assumed that appetite and the regulation of food intake were purely psychological phenomena, and that psychological investigation alone was needed to elucidate the problem of obesity. This is now known to be far from true. Much work has been and is being done on the role of various hypothalamic centres in regulating fat production and storage. A single injection of gold-thioglucose in animals will produce obesity due to selective destruction of cells in the ventro-medial area of the hypothalamus. There are no doubt other regulatory mechanisms. There are forms of obesity which are purely psychogenic, but they have to be distinguished more clearly from those that are not.

THE TREATMENT OF INTRACTABLE PAIN.

THE treatment of intractable pain in certain diseases and in the terminal stages of others almost always presents problems. J. S. Richardson,[1] in a symposium on the treatment of pain at the South African Medical Congress in 1957, presented a paper in which he reviewed the use of a number of the newer drugs in the treatment of intractable pain.

The adrenal steroid hormones are very useful for the pain in acute rheumatoid arthritis, provided that the difficulties associated with their use are always taken into account. The pain of acute gout which has failed to respond to the more usual measures will often react dramatically to cortisone, although there may be relapse when the cortisone is withdrawn; but this may be prevented by colchicine. Steroid hormones and corticotropin, in combination with alkylating agents such as triethylene melamine and nitrogen mustard, will often control intense pain where there are widespread metastases, and in Hodgkin's disease and follicular lymphoma. Adequate hæmatological control is important in these cases. Antimetabolite drugs such as 6-mercaptopurine, which has practically no toxicity, are useful in acute leukæmia, with or without cortisone or corticotropin. The sex hormones are useful in some forms of cancer. Destruction of the pituitary by implantation of radioactive gold or yttrium is being tried in several centres in cases of advanced cancer.

Deep X irradiation is of great value in controlling pain in some metastatic deposits in bone. Irradiation of the parotid gland in the early stages of acute parotitis is effective in controlling pain. This is particularly useful when the patient is in a severely debilitated state. Whatever other methods are used, sooner or later the use of analgesic drugs will play some part in the treatment of chronic pain. The value of mild analgesics is sometimes greater than is commonly realized. Richardson quotes H. K. Beecher, who showed that "10 gr. of acetylsalicylic acid was demonstrably more effective as an analgesic than 1/6 gr. of morphia or 1 gr. of codeine, all by mouth". The relief of anxiety is a first step in the management of chronic pain. To this end chlorpromazine has been used for some time, but large doses are required. About 150 milligrammes in 24 hours are required in doses of 25 milligrammes taken by mouth four times a day with 50 milligrammes at night. While chlorpromazine has itself no analgesic effect, its use lessens the quantity of analgesic required, besides improving the mental attitude of the patient.

Morphine or one of its substitutes will be required in many cases. Methorphinan ("Dromoran") is particularly useful, as it can be given effectively by mouth, and it causes less nausea than morphine. The respiratory depression which is produced by morphine, pethidine and "Dromoran" can be reduced by levallorphan tartrate in doses of approximately 0·5 to 1·5 milligrammes. For increasing the effectiveness of morphine and diminishing its ill effects in the control of intractable pain in the terminal stages, amiphenazole ("Daptazol") is useful, particularly when large doses of morphine (1·0 to 2·5 grains) are being used. Respiratory depression, narcosis and reduction in the cough reflex are said not to occur. While morphine or its substitutes will often have to be given towards the end, it is not always necessary to resort to injections immediately and thus abandon the great advantages of oral medication. The object, Richardson states, is to prevent the agonizing pain, but not necessarily to abolish pain entirely (which may result in severe clouding of consciousness alternating with periods of agony), so that our patients may die with all possible dignity as well as in peace.

[1] *South African M. J.*, 1958, 32:158 (February 8).

ARTIFICIAL INSEMINATION IN THE HUMAN.

THE urge to parenthood is one of the strongest drives and its denial one of the greatest deprivations. In Genesis, Rachel was barren and said to Jacob: "Give me children, or else I die." This feeling is echoed in the hearts of many women, and when the male partner is at fault the anguish is intensified for both. This private state of affairs is only too obvious to those interested in the subject, and to the physician, dedicated to the relief of physical and psychic illness and wishing to contribute to the sum of total human happiness, heterogenous insemination carried out with certain safeguards might seem the ready answer. Such a private solution, while it may satisfy, has obligations of a moral and legal nature extending to the rest of society, and involving the donor, the unborn child and the doctor. Since the matter lies in the field of sex and is emotionally charged, attitudes are as varied as religious creeds.

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Abstracts from Medical Literature.

OTO-RHINO-LARYNGOLOGY.

Serous Otitis Media.

M. M. AUSLANDER (*Arch. Otolaryng.*, January, 1958) reports on the treatment of serous *otitis media* with injectable trypsin. Many cases of secretory *otitis media* respond to simple management utilizing a single myringotomy *plus* Eustachian tube inflation. There are many which become protracted, requiring multiple treatments. The ætiology is not quite clearly known. There may be a readily inflatable Eustachian tube, yet the middle ear continues to fill. Since "Parenzyme" (a pure crystalline trypsin in sesame oil) had aided reduction of ecchymoses and œdema after rhinoplasty operations, it was thought that the same substance might be of value in serous *otitis media*. Following myringotomy and removal of exudate by suction together with Eustachian inflations, "Parenzyme" (containing 2·5 milligrammes of trypsin) was injected intramuscularly twice daily for five days. Of 106 patients, 89 responded favourably to one myringotomy and "Parenzyme" treatment. A second myringotomy was required in 17. All cases were completely free of fluid by the fifth day. The mechanism involved in trypsin therapy is not understood, but results were better with it than without it.

Prevention of Adverse Effects of Streptomycin on the Ear.

T. OZAKI (*Arch. Otolaryng.*, December, 1957) reports on the adverse after effects of streptomycin therapy in 39 tuberculous patients. If the audiogram shows any change, 100 milligrammes of vitamin B_1 should be administered intravenously each day. During this treatment audiograms are taken every 10 days. If improvement is shown, this treatment should be continued until there is complete recovery. To prevent recurrence, the treatment should be continued with injections twice a week for the first month, once a week for the second month, and two or three times in the third month. This treatment should cover a six-months' period and the number of treatments should be gradually reduced. Even when no improvement is evident, continued treatment may retard the development of symptoms. Of 29 patients so treated, there was complete recovery in 15, or improvement of 20 decibels or more in one or both ears, and subjective symptoms were eliminated. Tinnitus, however, was often obstinate, although apparently controllable if treated early. The dosage of streptomycin administered does not appear to be the sole factor in the adverse after-effects. There were more cases of adverse after-effects among patients receiving injections totalling 30 grammes or more. However, among patients who received only 10 grammes there were six cases, and even amongst those injected with two, five or six grammes there were some cases. There may thus be individual susceptibility. Streptomycin does not affect other organs, so may be presumed to have an affinity for the inner ear. At first a dose of one gramme of streptomycin administered daily for 60 days was the maximum dosage. Later, injections of one gramme twice a week enabled doses totalling over 60 grammes to be given. The symptoms of streptomycin sulphate appear three to six months after treatment. With dihydrostreptomycin injections the symptoms appear during the treatment. If the symptoms are detected in the early stage and treatment is given promptly, a complete cure is possible. However, it may be difficult to detect early effects. Otalgia, a feeling of obstruction or compression in the ear and tinnitus should be watched for. In cases of adverse effect the audiogram soon shows a lowering of perception of high-pitched sound and gradually that of low-pitched sounds. The author recommends that 100 milligrammes of vitamin B_1 should be injected intravenously immediately after each streptomycin injection, as a prophylactic measure. He claims that since this method was adopted there has been a steadily decreasing number of patients with impaired hearing.

Recurrent Nasal Polypi.

E. B. KORKIS (*Arch. Otolaryng.*, January, 1958) states that the main problem in the treatment of nasal polyposis is recurrence. The investigation and treatment of allergies have not proved entirely successful in preventing recurrences. Desensitizing and antihistamine drugs have proved disappointing. The value of cortisone remains uncertain. That recurrences are frequent after intranasal and transantral ethmoidectomy may be attributed to an inability to perform a thorough exenteration of the ethmoid labyrinth by use of these techniques. It is the author's practice to perform an external ethmoidectomy when recurrences are frequent. In a series of 70 such operations on 42 patients, primary healing of the wound occurred in all, and complications were negligible. The cases have been followed up for periods up to nine years. There have been three localized minor recurrences in 70 operations, and these were observed within the first three years after operation, but none was found after that period. A persistent postnasal discharge was reported by some patients, mainly allergic subjects, but this was occasionally attributable to low grade pan-sinus infection. Crusting after the operations was negligible, and alkaline nasal douching sufficed to control it. The cosmetic end results of all operations have been excellent.

Repair of the Facial Nerve.

K. KETTLE (*Arch. Otolaryng.*, January, 1958) discusses repair of the facial nerve in traumatic facial palsies. He states that Professor N. M. Dott, of Edinburgh University, has suggested a method of dealing with intracranial lesions of the facial nerve by means of a graft which by-passes the temporal bone to join the two sections of the facial nerve. The facial nerve is exposed at the cerebello-pontine angle by a unilateral cerebellar approach. The nerve is severed, and to its proximal end a nerve graft some 15 centimetres long is sutured. The free end of the graft is then brought out through the cranial opening and is passed along a tunnel beneath the mastoid process, between the sterno-mastoid and *splenius capitis* muscles, extending forward to the posterior surface of the parotid gland. Ninety days are allowed for nerve fibres to pass down along the graft to its distal end. The freshened end of the graft is now brought to the severed end of the peripheral segment of the facial nerve and sutured to it. The entire intrapetrous segment of the facial nerve is thus by-passed. Dott has reported operation in this manner on four patients. In two an 80% recovery ensued, and in the other two 50% recovery. The author regards Dott's operation as one of the greatest advances in facial nerve surgery, permitting restoration of function in cases where intrapetrous operation is impracticable, such as in lesions proximal to the geniculate ganglion, or when the temporal bone is destroyed.

PATHOLOGY.

Transaminase and Hepatic Disease.

R. A. DONATO (*Am. J. Clin. Path.*, October, 1957) has correlated the transaminase activity of serum with the findings of other liver function tests, including liver biopsy. He found that the level of glutamic oxalacetic transaminase activity in the serum is a much more sensitive indicator of minimal to moderate liver damage than any of the usual tests of liver function. There is also a correlation between the level of transaminase activity and the extent of hepatocellular damage.

Idiopathic Familial Oxalosis.

D. L. EDWARDS (*Arch. Path.*, November, 1957) has reported an instance of the familial occurrence of a rare disturbance of metabolism. Two adult male siblings were found to have parenchymal deposits of calcium oxalate crystals in the kidneys, leading to renal damage sufficient to cause death. Similar crystals were found in the testes of one sibling and in the myocardium of both. The clinical and pathological features of the cases are discussed.

Experimental Iron Overloading.

E. B. BROWN *et alii* (*J. Lab. & Clin. Med.*, December, 1957) have studied the effects of iron overload in dogs for periods of four to seven years. Four animals were given iron intravenously as saccharated iron oxide; two were transfused with whole blood. The total dose was 0·5 or 1·0 gramme of iron per kilogram of body weight. No evidence of cirrhosis could be found by liver function tests or by histological sections at biopsy or autopsy. There was no diabetes or pancreatic fibrosis. Heavy iron deposits were found predominantly in reticulo-endothelial cells. There was no evidence of any fibrous tissue reaction. Blindness developed in all of the animals, the eye lesions resembling those of *retinitis pigmentosa*. With this exception the

tissue iron overload was remarkably well tolerated. Hæmatological changes were plethora (in the transfused dogs) and serum iron elevations to values 20 times normal. From the chemical estimation of the tissue iron in biopsy and autopsy specimens there is no evidence that redistribution of the iron load occurred. From 37% to 73% of the administered iron could be accounted for.

Inflammation in Alloxan Diabetes.

M. K. SCHAUBLE AND R. D. BAKER (*Arch. Path.*, November, 1957) studied the inflammatory response to *Staphylococcus pyogenes* and the spores of *Rhizopus oryzæ* in three groups of rabbits: (i) those in the acute toxic phase of alloxan diabetes; (ii) those in the chronic phase of alloxan diabetes; (iii) normal controls. There was a good inflammatory response in each group, but in both infections proliferation of the organism was greater in acutely toxic alloxan diabetic animals. In the case of *R. oryzæ*, vascular invasion occurred in animals in the acutely toxic stage. The authors conclude that the inflammatory response is satisfactory in the acute diabetic state, and that the enhanced invasiveness of the organisms is due to some other factor.

Chronic Cystic Mastitis.

P. T. SLOSS, W. A. BENNETT AND O. T. CLAGETT (*Am. J. Path.*, November-December, 1957) report the results of gross and microscopic examination of the breasts of 100 female subjects seen at necropsy. All these breasts had been considered as entirely normal on clinical examination. A high incidence of the changes considered to be part of the complex of chronic cystic mastitis was disclosed. Therefore, the authors conclude that the mere qualitative presence of adenosis, apocrine epithelium and intraductal epithelial hyperplasia in the breasts of women is insufficient to warrant such tissue being considered as diseased.

Excessive Oozing After Surgery.

M. B. ZUCKER *et alii* (*J. Lab. & Clin. Med.*, December, 1957) have carried out a study of blood clotting factors and fibrinolysis in 78 patients before, during and after major surgery. Fifteen patients oozed excessive amounts of blood during surgery. Oozing could not be ascribed to massive blood replacement, and in none of the oozing patients was the platelet count below 100,000 per cubic millimetre. Plasma citrate concentration bore no relationship to the occurrence of oozing. Fibrinolytic activity of the euglobulin fraction was found during or shortly after surgery in nine of the 15 oozing patients. Active whole clot lysis was observed in six cases. Fourteen of the 63 patients who did not show excessive oozing had euglobulin fibrinolytic activity and five had whole clot lysis. Fibrinogen concentration fell below 100 milligrammes per 100 millilitres in only three of the oozing patients, two of whom were undergoing hepatic lobectomy. Decreases in prothrombin and factor VII concentrations during surgery occurred with equal frequency among both groups o patients, whereas decreases in factor V occurred more frequently in oozing patients. Values below 50% of normal were observed only in patients undergoing hepatic lobectomy. All but one of 14 oozing patients showed a change in at least one of the factors measured. Three had poor prothrombin consumption before surgery, three had prothrombin or factor V concentrations below 70%, and the remaining seven showed fibrinolytic activity *plus* some other change. The correlation between oozing and the occurrence of multiple changes was statistically significant.

Significance of Blood Group Conflicts in Spontaneous Abortion.

C. MCNEIL *et alii* (*Am. J. Clin. Path.*, November, 1957), in a preliminary communication, have published their findings concerning the blood groups of couples in whom the wife suffers from habitual abortion. They found that these couples could be classified into those in which a group A or B father was mated with a group O mother; those which manifested marked irregularity in the salivary secretion of A, B and H factors; those which were characterized by both these features. The significance of these findings is discussed.

THERAPEUTICS.

Hypercalcæmia and Hypercalcuria in Disseminated Malignant Disease.

W. P. L. MYERS (*Cancer*, January-February, 1958) has treated 11 patients with hypercalcæmia and hypercalcuria secondary to widespread cancer, with cortisone, prednisone, or hydrocortisone. The types of neoplasms included carcinoma of the kidney and carcinoma of breast, lymphosarcoma, multiple myeloma, metastatic adenocarcinoma of unknown primary origin and rhabdomyosarcoma. There was complete reversal of the calcium changes in five patients, a partial reversal in two, and no response in four. A response usually occurred within two weeks and longer periods of treatment, in general, were ineffective. Inhibition of tumour growth rate appeared to be the mechanism in some cases, in others it was obscure.

Myasthenia Gravis.

R. S. SCHWAB *et alii* (*J.A.M.A.*, October 12, 1957) describe the treatment of *myasthenia gravis*. Injections of neostigmine or 15-milligramme tablets of neostigmine given by mouth relieve the symptoms for one to three hours. Since 1953 "Mestinon" bromide has been in use, and more recently prolonged action tablets of "Mestinon" bromide of 30 or 60 milligrammes. It was found that with a dose of 180 milligrammes these tablets controlled the symptoms satisfactorily for two and a half times as long as 60 milligrammes of ordinary "Mestinon". The prolonged action tablets contained a core of inert material into which the "Mestinon" is absorbed. Of 109 patients, 82 preferred to take 90 to 180 milligrammes of long-acting "Mestinon" every four to six hours, rather than 30 to 60 milligrammes of ordinary "Mestinon" every two or three hours. A further study with slow release tablets of neostigmine bromide showed that a 45 milligrammes slow release tablet had a more prolonged effect than a 15 milligrammes tablet of ordinary neostigmine bromide, and 54 out of 85 patients preferred this treatment. The benefit of the slow release tablets was that they had to be taken once in six hours instead of every two to three hours, as was the case with ordinary neostigmine. In some patients the 45 milligrammes tablet caused diarrhœa and cramps and it was found that a half strength tablet was sufficient. Great care was needed in adjusting the dose to different patients, and a number preferred to continue with their former doses of ordinary neostigmine or "Mestinon" bromide.

Promazine Hydrochloride.

THE COUNCIL OF DRUGS OF THE AMERICAN MEDICAL ASSOCIATION (*J.A.M.A.*, October 12, 1957) reports 18 cases of blood dyscrasia apparently associated with promazine ("Sparine") hydrochloride. In some cases other drugs such as chlorpromazine hydrochloride had also been given to the patients. Depression of granulocytes was marked in every case reported and bone marrow studies in some cases indicated a depression of other cells as well. There were four deaths in this series. In those cases which were suspected, early cessation of the drug and appropriate treatment were usually followed by recovery. Initial symptoms such as sore throat, fever or malaise should be sought. Interim blood counts are not sufficient, as the condition can develop between counts suddenly. The Council advises that promazine should be used only when other drugs do not suffice.

Amœbiasis.

J. M. BUSTAMENTE Y RIVERO of Peru (*J.A.M.A.*, October 19, 1957) reports on the treatment of acute and chronic amœbiasis with "Camoform" (biallylamicol). "Camoform" has been found effective in-vitro against *Entamœba histolytica*. In a previous paper the author had reported rapid therapeutic response and cure in 82 out of 85 patients with acute amœbic colitis. He also notes favourable reports on this drug from several other countries. In his present paper he reports on 39 patients with proved chronic amœbic dysentery, and on one patient with pulmonary amœbiasis. "Camoform" was given in doses of 250 or 500 milligrammes three times a day with meals for 15 days. All patients became free from symptoms after one course of treatment, and *E. histolytica* could no longer be found. Other organisms, including *Giardia lamblia* and *Trichomonas hominis*, were significantly reduced. The patient with pulmonary amœbiasis was treated with 750 milligrammes of "Camoform" daily for six days with four injections of 400,000 units of penicillin. The symptoms and signs disappeared within a week, and the patient was still symptom free 10 weeks later. The only toxic symptoms reported were, in a few instances, nausea, vomiting and abdominal distress, but these were not sufficient to prevent continuance of treatment.

British Medical Association.

NEW SOUTH WALES BRANCH: ANNUAL MEETING.

THE annual meeting of the New South Wales Branch of the British Medical Association was held at the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, on March 27, 1958, DR. G. L. HOWE, the President, in the chair.

ANNUAL REPORT OF THE COUNCIL.

The annual report of the Council was taken as read and received on the motion of Dr. T. Y. Nelson, seconded by Dr. M. S. Alexander. The report was commented on by Dr. Nelson, Dr. Edgar Thomson, the President, Dr. Lindon Wing and also Dr. G. J. M. Saxby, who asked that in future the annual report of the Council should be circulated in advance among members, so that they might have a chance to study it. The report was adopted on the motion of Dr. T. Y. Nelson, seconded by Dr. M. S. Alexander.

The annual report is as follows.

The Council presents the following report on the work of the Branch for the year ended March 27, 1958.

Membership.

The membership of the Branch is now 4,098, as against 4014 at the date of the last report. The additions have included 161 elections, re-elections and resumptions, and 130 removals into the area of the Branch; while the losses have included 30 by resignation, 103 removals out of the area of the Branch, 37 by default in payment of subscription, and 37 by death. The losses by death were as follows: Dr. R. Hilliard, Dr. A. W. Holmes à Court, Dr. J. Harris, Dr. G. A. Thompson, Dr. E. B. L. Fitzpatrick, Dr. J. A. S. Brown, Dr. G. A. W. Johnston, Dr. H. L. Spearman, Dr. E. Ludowici, Dr. R. Nihill, Dr. Theodora M. England, Dr. D. A. Hughes, Dr. E. F. Coyle, Dr. E. F. Fisher, Dr. Dorothy M. McClemens, Dr. A. E. D. Clark, Dr. G. H. Hair, Dr. E. S. Morris, Dr. M. Archdall, Dr. A. H. Williams, Dr. T. W. Miles, Dr. R. M. Allport, Dr. Maida E. W. B. Hall, Dr. Ethel Byrne, Dr. L. T. Allsop, Dr. Weeks White, Dr. W. J. Binns, Dr. Phyllis M. Anderson, Dr. M. R. Flynn, Dr. A. L. Caselberg, Dr. W. Evans, Dr. G. B. White, Dr. F. J. Reynolds, Dr. C. W. Randall, Dr. E. M. C. Friedlander, Dr. C. L. Chapman, Dr. R. M. Thomson.

Obituary.

In the deaths of the following the medical profession has suffered a great loss.

Mervyn Archdall.

Dr. Mervyn Archdall, second Editor of THE MEDICAL JOURNAL OF AUSTRALIA, died on September 6, 1957, six days after his official retirement from the editorial chair, at the age of 73.

In 1930 Dr. Mervyn Archdall succeeded Dr. H. W. Armit as Editor, having worked as Assistant Editor the previous eight years. During his 27 years as Editor he raised the standard of THE MEDICAL JOURNAL OF AUSTRALIA to such a height as to command world-wide respect.

In recognition of his services to the medical profession in Australia the Federal Council bestowed upon him its Gold Medal, the highest award the Association in Australia can confer upon any of its members. This award was made just a few days before his death.

The deep sympathy of the Branch is extended to Mrs. Archdall.

A. W. Holmes à Court.

Dr. Alan Worsley Holmes à Court, who died on April 16, 1957, was a member of the Council for the years 1925-1929 and 1931-1935, and was President during the year 1933. He was also a Past President of the Royal Australasian College of Physicians.

The sincere sympathy of the Branch is extended to his family.

Phyllis M. Anderson.

Dr. Phyllis Margery Anderson, who died on November 29, 1957, was one who took a great interest in the welfare of women members of the profession. During a period of three years, from March, 1951, to March, 1954, she represented the women members of the New South Wales Branch of the British Medical Association on the Council. Prior to that she had been the Honorary Secretary of the Section of Pathology for five years. In addition she had been a member of the Standing Committee of Convocation.

Allan S. Walker.

For many years Dr. Allan Seymour Walker, who died on January 8, 1958, practised as a specialist physician in Sydney, but he retired from this field to become Editor of the Australian Medical War History, 1939-1945.

During the years 1935-1940 he was a member of he Council of the New South Wales Branch of the British Medical Association.

The sympathy of the Branch is extended to Mrs. Walker and family.

Weeks White.

The late Dr. Weeks White, who died on November 19, 1957, at the age of 44 years, was one of the select band of country general practitioners who have served their profession by becoming members of Council.

Practising at Leeton, he became a member of Council following the election in 1957. He was a regular attendant at the meetings of Council and its committees.

The deep sympathy of the Branch is extended to Mrs. White and family.

Meetings.

Ten ordinary general meetings of the Branch (including the annual general meeting), two extraordinary general meetings of the Branch, and ten clinical meetings were held. The average attendance was 58.

Eight ordinary general meetings were held in conjunction with meetings of the special groups, viz.: April 24, with the Section of Medicine, the Section of Neurology, Psychiatry and Neuro-Surgery and the Section of Pædiatrics; June 27, with the Section of Surgery, the Section of Radiology and the Dermatological Association of Australia (British Medical Association); July 25, with the Orthopædic Group (British Medical Association), the Section of Pædiatrics and the Section of Pathology; August 29, with the Section of Pædiatrics, the Section of Urology and the Section of Pathology; September 26, with the Section of Anæsthesia and the Section of Medicine; October 31, with the Section of Occupational Medicine, the Orthopædic Group (British Medical Association) and the Section of Radiology; November 30, with the Section of Pædiatrics; December 12, with the Section of Surgery, the Section of Obstetrics and Gynæcology, and the Section of Neurology, Psychiatry and Neuro-Surgery. Sixteen papers were presented at these meetings.

The clinical meetings were held at the Rachel Forster Hospital for Women and Children, Royal North Shore Hospital, Royal Prince Alfred Hospital, Royal Alexandra Hospital for Children, Saint Vincent's Hospital, Lewisham Hospital, Sydney Hospital, The Women's Hospital, Crown Street, The Saint George Hospital and the Psychiatric Clinic at Broughton Hall.

Three medical films were shown at the ordinary general meeting on May 30, and at the extraordinary general meeting on December 12 the Articles of Association (Articles 37, 40 (i), 53, 56) relating to vice-presidents were amended so as to provide that there may be more than one vice-president, and that a vice-president shall have and enjoy such duties, powers and privileges as shall be determined from time to time by the by-laws, but shall not be a member of Council. At the request of more than twenty members, the extraordinary general meeting on February 12 was convened to consider the question of medical education, and it was decided to appoint a committee to draw up a report for presentation to Council at a future date.

The ordinary general meeting on Saturday, November 30, and Sunday, December 1, was held in Leura, it being the sixth meeting of the Branch to be held in a country town. There were 25 members present and two papers were read, these being presented by the Section of Pædiatrics. In addition to a scientific programme, social functions were arranged by the Blue Mountains Medical Association. The Council extends its grateful thanks to the Blue Mountains Medical Association for its assistance in the organization of the meeting.

An invitation was extended to the fifth and sixth year medical students of the University of Sydney to attend ordinary general meetings, and to sixth year medical students to attend clinical meetings of the Branch.

Representatives.

The Branch was represented as follows:

1. Council of the British Medical Association: Professor B. W. Windeyer.
2. Annual Representative Meeting, British Medical Association, Newcastle-on-Tyne, July, 1957: Dr. M. H. Elliot-Smith, Dr. J. P. Lyttle and Dr. J. A. Paul.
3. Post-Graduate Committee in Medicine, The University of Sydney: Dr. S. R. Dawes, Dr. K. S. Jones and Dr. E. F. Thomson.

4. Associated Council for Health: Dr. D. G. Hamilton and Dr. L. W. Wing.
5. New South Wales Bush Nursing Association: Dr. G. L. Howe.
6. New South Wales Institute of Dietitians: Dr. K. S. Harrison.
7. State Medical Planning Committee: Dr. M. S. Alexander, O.B.E.
8. Florence Nightingale Memorial Committee of Australia: Dr. Mary Puckey.
9. Medical Appointments Advisory Committee: Dr. T. Y Nelson.
10. Old People's Welfare Council of New South Wales: Dr. G. L. Howe.
11. City of Sydney Youth Welfare Advisory Committee: Dr. G. L. Howe.
12. Committee for the Placement of Resident Medical Officers: Dr. T. Y. Nelson.
13. New South Wales Institute of Hospital Almoners: Dr. R. A. R. Green.
14. The Ophthalmic Association Ltd.: Dr. E. V. Waddy Pockley.
15. Federal Council of the British Medical Association in Australia: Dr. A. J. Murray, O.B.E., Dr. R. H. Macdonald, O.B.E., Dr. W. F. Simmons and Dr. E. F. Thomson.
16. New South Wales Association for Mental Health: Dr. A. T. Edwards.
17. New South Wales Examining Council in Medical Technology (Hospitals Commission of New South Wales): Dr. E. F. Thomson and Dr. F. S. Hansman.
18. Standards Association of Australia: (i) Safety Standards Co-ordinating Committee, Dr. W. E. George; (ii) Institutional Supplies Committee, Dr. S. W. G. Ratcliff; (iii) Sectional Committee on Interior Illumination of Buildings, Dr. J. Davis; (iv) Committee of Standards of Laboratory Glassware and Volumetric Glassware, Dr. F. S. Hansman; (v) New South Wales Committee on Protective Occupational Clothing, Dr. J. H. Blakemore; (vi) Paint and Varnish Sub-Committee No. 8, Dr. J. H. Blakemore; (vii) New South Wales Committee on Eye Protection, Dr. J. Davis; (viii) Sectional Committee on Measuring Cups and Spoons, Dr. W. W. Ingram; (ix) New South Wales Committee on Industrial Respiratory Protective Devices, Dr. W. E. George.
19. Royal Flying Doctor Service of Australia, New South Wales Section: Dr. George Bell, O.B.E.
20. New South Wales College of Nursing: Dr. E. F. Thomson.
21. Australasian Medical Publishing Co. Ltd.: Dr. W. F. Simmons, Dr. W. L. Calov, Professor L. F. Dods, M.V.O.
22. Hospitals Contribution Fund of New South Wales: Dr. Hugh Hunter.
23. St. John Ambulance Association: Dr. G. L. Howe.
24. Special Departmental Committee for Investigation of Maternal Deaths: Dr. E. A. Tivey. Alternate Representative: Dr. M. H. Elliot-Smith.
25. Medical Officers' Relief Fund (Federal). Local Committee of Management for New South Wales: Dr. A. M. McIntosh, Dr. A. J. Murray, O.B.E., and Dr. R. H. Macdonald, O.B.E.
26. New South Wales Medical Board: Dr. J. R. Ryan.
27. Medical Finance Limited, Board of Directors: Dr. E. A. Tivey, Dr. A. C. Thomas, Dr. George Bell, O.B.E., and Dr. G. C. Halliday.
28. Co-ordinating Council of the Physically Handicapped: Dr. R. A. R. Green.
29. Road Safety Council of New South Wales: Dr. G. L. Howe.
30. Federal Medical War Relief Fund, Local Committee for Management: Dr. R. H. Macdonald, O.B.E., Dr. A. C. Thomas and Dr. A. J. Murray, O.B.E.
31. National Association for the Prevention of Tuberculosis in Australia (New South Wales Division): Dr. W. Cotter B. Harvey.
32. New South Wales State Cancer Council: Dr. B. T. Edye, C.B.E.
33. Department of Motor Transport (Committee to consider the question of the adoption of chemical tests of body fluids to determine whether a driver is under the influence of alcohol): Dr. F. S. Hansman.
34. Medico-Pharmaceutical Liaison Committee: Dr. K. S. Jones, Dr. G. L. Howe and Dr. W. F. Simmons.
35. Department of Public Health, Poisons Advisory Committee: Dr. A. W. Morrow, D.S.O.
36. National Health Service. Pensioner Medical Service Committee of Inquiry: Dr. M. S. Alexander, O.B.E., Dr. B. A. Cook, Dr. A. W. Morrow, D.S.O., and Dr. A C. Thomas.
37. State Medical Advisory Committee: Dr. E. F. Thomson. Alternate Representative: Dr. J. G. Hunter.
38. Fluoridation of Drinking Water. Department of Public Health Advisory Committee: Dr. D. G. Hamilton.
39. New South Wales Association of Medical Records Librarians Advisory Committee: Dr. T. Y. Nelson.
40. Board of Optometrical Registration: Dr. J. Davis.

Council.

(*a*) The attendance of members of the Council and of the standing committees was as set out in the accompanying table.

(*b*) The representatives of the Local Associations of members appointed on the invitation of the Council to attend the regular quarterly meetings of the Council were as follows: Dr. A. W. Raymond (Blue Mountains), Dr. A. B. Hogan (Border), Dr. W. A. Hillman (Brisbane Water District), Dr. L. Abramovich (Canterbury-Bankstown), Dr. E. J. Egan (Central Northern), Dr. M. V. Mutton (Central Southern), Dr. G. N. M. Aitkens (Central Western), Dr. H. E. Masters (Eastern District), Dr. D. B. Dunn (Eastern Suburbs), Dr. W. P. H. Dakin (Far South Coast and Tablelands), Dr. L. O. Rutherford (Hunter Valley), Dr. K. W. Alexander (Illawarra Suburbs), Dr. C. H. Anderson (Kuring-gai District), Dr. P. K. Bell (Nepean Hawkesbury), Dr. S. Shineberg (Northern District), Dr. J. Gribben (North Eastern), Dr. H. G. Rich (South Eastern), Dr. J. G. O'Neill (South Sydney), Dr. R. G. Bligh (Warringah District), Dr. S. A. Bonnette (Western Suburbs).

Library.

Dr. E. F. Thomson was appointed to the position of Honorary Librarian.

Visitors to the Library	7,512
Books lent to members	1,160
Journals lent to members	5,170
Books added to the Library	238
Journals added to the Library	8

Increases in the above figures, by comparison with those for last year, show that the Library is being used to a greater extent each year, and further interest in the development of the Library's resources is evidenced by the number of donations of periodical material, books, etc., which have been made during the past twelve months.

Due to the rapid growth of the Library the Council decided to make additional space available, and the conversion of the Council room into an extra storeroom and workroom was completed last year.

The increasing use of the Library has necessitated increase in staff and this, together with the increase in the number and price of periodicals and books, has resulted in increased library expenditure.

Details of the amount expended on the Library for the year ended December 31, 1957, are as follows:

	£	s.	d.
Salaries	3,188	11	3
Subscriptions to journals ..	1,131	11	9
Books	676	1	10
Binding	396	14	10
Photo copying equipment ..	44	7	7
	£5,437	7	3

This amount absorbed 13% of the net subscriptions received.

It is the wish of the Council to acknowledge with grateful thanks donations from the following: The Editor, THE MEDICAL JOURNAL OF AUSTRALIA; Abbott Laboratories; American College of Surgeons; Dr. D. B. Arnott; Dr. B. J. Basil-Jones; Dr. J. Cooper Booth; British Empire Cancer Campaign; British Medical Association, Victorian Branch; Professor A. N. St. G. H. Burkitt; Dr. B. P. C. Cahill; Dr. Grace J. Browne; Dr. D. G. Carruthers; Canadian Medical Association; Dr. J. N. Chesterman; Ciba Company Proprietary Limited; College of General Practitioners; Commonwealth of Australia, Department of Health; John Crerar Library, Chicago; Editorial Board, *Journal of Pediatrics*; Mrs. John Foley; Dr. B. E. Frecker; Dr. E. H. Freidman; the French Consulate on behalf of the French Government; Geigy Australasia Proprietary Limited; Dr. J. H. Halliday; Harker Stagg Limited, London; Dr. Margaret H. Harper; Mrs. E. S. Holloway; Dr. F. A. Innes-Brown; the Japanese Embassy, Canberra; Dr. A. I. Lane; Dr. F. A. E. Lawes; Dr. D. G. Maitland; May and Baker (Australia) Proprietary Limited; Mayo Clinic; Medical and Chirurgical Faculty of the State of Maryland Library; Metropolitan Life Insurance Company, New York; National Institute of Health Library, Bethesda, Maryland; National Nephrosis Foundation, Inc.; New South Wales Department

ATTENDANCE AT COUNCIL AND STANDING COMMITTEE MEETINGS.

	Council.	Committees.					
		Executive and Finance.	Organization and Science.	Medical Politics.	Hospitals.	Ethics.	Public Relations.
ALEXANDER, M. S.	14	15	—	—	—	1	—
BROWN, D. A.	8	—	—	—	—	—	5
COBLEY, J. F. C. C.	13	—	3	—	6	—	—
COOK, B. A.	11	—	—	—	3	—	7
HAMILTON, D. G.	14	—	6	—	5	—	—
HOWE, G. L.[1] President	11	11	2	4	2	1	2
ISBISTER, CLAIR	12	—	4	—	1	—	5
JONES, K. S.	15	—	—	9	—	—	7
KENNY, P. J.	11	—	—	—	5	—	—
LAVERTY, C. R. M.[2]	6	—	—	—	—	1	—
MACDONALD, R. H.	15	13	—	—	—	2	—
MONAHAN, B. W.	12	—	—	11	—	—	—
MORROW, A. W.[3] President-Elect	12	8	2	6	1	1	4
MURRAY, A. J.	15	12	—	—	—	3	—
NELSON, T. Y. Honorary Secretary	13	12	6	11	6	3	5
RAWLE, K. C. T.	6	—	—	—	5	—	—
SIMMONS, W. F. Honorary Treasurer	16	13	4	11	4	1	8
SPEIGHT, R. J. J.	14	—	—	9	—	—	—
STUCKEY, E. S.[4]	9	—	—	10	—	—	—
THOMSON, E. F. Past President	15	13	6	—	—	2	6
TOMLINSON, P. A.	14	—	—	—	4	—	—
WARDEN, D. A.	12	—	—	12	—	—	—
WHITE, WEEKS[5]	7	—	—	—	—	—	—
WILLIS, H. H.	13	—	—	—	—	3	—
WING, L. W.[6]	5	—	—	9	—	—	—
Meetings held	16	15	6	12	6	3	8

[1] Leave of Absence—2 months from April 10, 1957.
[2] Leave of Absence—5 months from April 9, 1957.
[3] Leave of Absence—3 months from September, 1957.
[4] Leave of Absence—January 6, 1958, to March 27, 1958.
[5] Died, November 19, 1957.
[6] Elected December 10, 1957.

of Health; New South Wales State Cancer Council; Pfizer Proprietary Limited; Post-Graduate Committee in Medicine, University of Sydney; Queensland Institute of Medical Research; Dr. Norman H. Rose; Dr. Howard Rusk (U.S.A.); Royal Australasian College of Physicians; Dr. Margery Scott-Young; Dr. Eva Shipton; Dr. R. J. Silverton; Dr. K. W. Starr; Dr. F. G. N. Stephens; Dr. A. C. Thomas; Dr. E. F. Thomson; United States Information Library; University of Melbourne; Vanderbilt University School of Medicine Library, Tennessee; Dr. M. Naomi Wing; Dr. William Wood; the College of Radiologists (Australia and New Zealand); the Dermatological Association of Australia (B.M.A.); the Oto-Laryngological Society of Australia, New South Wales Section (British Medical Association); the Section of Medicine; the Section of Obstetrics and Gynæcology.

Affiliated Local Associations of Members.

Blue Mountains (affiliated 1944): *Chairman*, Dr. L. Bamber; *Honorary Secretary*, Dr. N. Larkins. Membership 28. Six meetings were held.

Border (affiliated 1908): *Chairman*, Dr. G. Wearing Smith; *Honorary Secretary*, Dr. R. S. Hayter. Membership 21. Two meetings were held.

Brisbane Water District (affiliated 1948): *Chairman*: Dr. Z. J. Caska; *Honorary Secretary*, Dr. W. A. Hillman. Membership 22. Four meetings were held.

Broken Hill (affiliated 1942): *Honorary Secretary*, Dr. Franziska Schlink.

Canterbury-Bankstown (affiliated 1930): *Chairman*, Dr. D. J. Law; *Honorary Secretary*, Dr. A. Rumore. Membership 87. Seven meetings were held.

Central Northern (affiliated 1910): *Chairman*, Dr. R. U. Bourke; *Honorary Secretary*, Dr. J. N. Walker.

Central Southern (affiliated 1909): *Chairman*, Dr. L. H. McCaffery; *Honorary Secretary*, Dr. R. J. Hoy. Membership 68. Three meetings were held.

Central Western (affiliated 1910): *Chairman*, Dr. L. P. Blashki; *Honorary Secretary*, Dr. K. S. M. Brown. Membership 75. Two meetings were held.

Eastern District (affiliated 1913): *Chairman*, Dr. J. R. O. Roger; *Honorary Secretary*, Dr. H. E. Masters. Membership 45. Two meetings were held.

Eastern Suburbs (affiliated 1911): *Chairman*, Dr. E. A. Jackson; *Honorary Secretary*, Dr. Z. S. Freeman. Membership 172. Four meetings were held.

Far South Coast and Tablelands (affiliated 1935): *Chairman*, Dr. W. P. H. Dakin; Honorary Secretary, Dr. J. D. F. O'Keefe. Membership 24. Three meetings were held.

Hunter Valley (affiliated 1947): *Chairman*, Dr. P. W. Gill; *Honorary Secretary*, Dr. A. J. R. Clarke. Membership 51. Four meetings were held.

Illawarra Suburbs (affiliated 1913): *Chairman*, Dr. M. C. Seton; *Honorary Secretary*, Dr. K. W. Alexander. Membership 84. Four meetings were held.

Kuring-gai District (affiliated 1929): *Chairman*, Dr. W. L. Morris; *Honorary Secretary*, Dr. R. B. Geeves. Membership 115. Four meetings were held.

Northern District (affiliated 1911): *Chairman*, Dr. R. S. Irwin; *Honorary Secretary*, Dr. J. H. Priestley. Membership 90. Three meetings were held.

North Eastern (affiliated 1913): *Chairman*, Dr. F. L. Nicholl; *Honorary Secretary*, Dr. N. J. Rogers. Membership 71. Three meetings were held.

Nepean Hawkesbury (affiliated 1957): *Honorary Secretary*, Dr. J. J. Bain.

South Eastern (affiliated 1914): *Chairman*, Dr. M. C. McKinnon; *Honorary Secretary*, Dr. G. R. James. Membership 77. Three meetings were held.

Southern District (affiliated 1909): *Chairman*, Dr. A. F. Smith; *Honorary Secretary*, Dr. B. W. Harding. Membership 88. Three meetings were held.

South Sydney (affiliated 1909): *Chairman*, Dr. J. G. O'Neill; *Honorary Secretary*, Dr. A. R. G. Gordon. Membership 16. One meeting was held.

Warringah District (affiliated 1929): *Chairman*, Dr. R. T. C. Hughes; *Honorary Secretary*, Dr. D. C. Williams. Membership 197. Four meetings were held.

Western (affiliated 1908): *Chairman*, Dr. F. G. Steele; *Honorary Secretary*, Dr. S. R. Dawes. Membership 115. Three meetings were held.

Western Suburbs (affiliated 1908): *Chairman*, Dr. L. H. Hughes; *Honorary Secretary*, Dr. Warren Smith. Membership 137. Three meetings were held.

Annual Meeting of Delegates.

The forty-fourth annual meeting of delegates of the affiliated local associations of members with the Council was held on Friday, October 4, 1957.

The delegates present at the meetings were as follows: Blue Mountains, Dr. A. W. Raymond; Border, Dr. M. M. Ramsden; Broken Hill, Dr. H. J. P. McMeekin; Brisbane Water, Dr. W. A. Hillman; Canterbury-Bankstown, Dr. L. Abramovich; Central Northern, Dr. L. N. Ferrari; Central Western, Dr. G. N. M. Aitkens; Eastern District, Dr. H. E. Masters; Eastern Suburbs, Dr. L. H. McMahon; Hunter Valley, Dr. L. O. Rutherford; Illawarra Suburbs, Dr. K. W. Alexander; Kuring-gai District, Dr. C. Warburton; Nepean

Hawkesbury, Dr. P. K. Bell; Northern District, Dr. R. J. Jackson; North Eastern, Dr. J. Gribben; Southern District, Dr. J. S. Storey; South Eastern, Dr. N. J. D. Powrie; South Sydney, Dr. G. Grant; Warringah District, Dr. R. G. Bligh; Western, Dr. G. B. Downes; Western Suburbs, Dr. S. A. Bonnette.

Special Groups for the Study of Special Branches of Medical Knowledge.

Allergy (inaugurated 1947): *Chairman*, Dr. S. E. L. Stening; *Honorary Secretary*, Dr. D. O. Cross. Membership 16. Five meetings were held.

Anæsthesia (inaugurated 1934): *Chairman*, Dr. R. B. Speirs; *Honorary Secretary*, Dr. C. A. Sara. Membership 83. Three meetings were held, one in conjunction with a meeting of the Branch.

Medicine (inaugurated 1924): *Chairman*, Dr. F. A. E. Lawes; *Honorary Secretary*, Dr. E. L. Davis. Membership 82. Six meetings were held, two in conjunction with meetings of the Branch.

Neurology, Psychiatry and Neurosurgery (inaugurated 1924): *Chairman*, Dr. Irene Sebire; *Honorary Secretary*, Dr. C. Radeski. Membership 96. Ten meetings were held, two with meetings of the Branch.

Obstetrics and Gynæcology (inaugurated 1925): *Chairman*, Dr. J. N. Chesterman; *Honorary Secretary*, Dr. A. S. B. Studdy. Membership 104. Six meetings were held, one in conjunction with a meeting of the Branch.

Occupational Medicine (inaugurated 1952): *Chairman*, Dr. G. C. Smith; *Honorary Secretary*, Dr. D. G. Noble. Membership 27. Four meetings were held, one in conjunction with a meeting of the Branch.

Orthopædic Group (British Medical Association) (inaugurated 1923): *Chairman*, Dr. C. C. McKellar; *Honorary Secretary*, Dr. K. R. Daymond. Membership 36. Seven meetings were held, two with meetings of the Branch.

The Oto-Laryngological Society of Australia, New South Wales Section (formerly The Oto-Rhino-Laryngological Society of New South Wales) (inaugurated 1924): *Chairman*, Dr. A. B. K. Watkins; *Honorary Secretary*, Dr. V. D. Bear. Membership 48. Six meetings were held.

Pædiatrics (inaugurated 1924): *Chairman*, Dr. S. P. Bellmaine; *Honorary Secretary*, Dr. D. Kerr Grant. Membership 105. Seven meetings were held, four in conjunction with meetings of the Branch.

Pathology (inaugurated 1924): *Chairman*, Dr. J. P. E. O'Brien; *Honorary Secretary*, Dr. A. A. Palmer. Membership 95. Seven meetings were held, two in conjunction with meetings of the Branch.

Radiology (inaugurated 1926): *Chairman*, Dr. D. G. Maitland; *Honorary Secretary*, Dr. B. E. Frecker. Membership 104. Ten meetings were held, two in conjunction with meetings of the Branch.

Surgery (inaugurated 1925): *Chairman*, Dr. J. Steigrad; *Honorary Secretary*, Dr. K. S. Jones. Membership 42. Four meetings were held, two in conjunction with meetings of the Branch.

Urology (inaugurated 1940): *Chairman*, Dr. D. C. Trainor; *Honorary Secretary*, Dr. H. G. Cummine. One meeting was held in conjunction with a meeting of the Branch.

Congratulations.

Congratulations were extended to Sir Ronald Grieve, K.B., Dr. B. T. Edye, C.B.E., and to Dr. T. J. Biggs, O.B.E., on the honours conferred upon them by Her Majesty the Queen.

Federal Council of the British Medical Association in Australia.

The Federal Council of the British Medical Association in Australia met in Adelaide on August 30-September 2, 1957, and in Hobart on February 26-28, 1958.

On both occasions the Branch was represented by Dr. A. J. Murray, Dr. W. F. Simmons, Dr. R. H. Macdonald and Dr. E. F. Thomson.

British Medical Association.

Election of Members of the Council by Branches not in Great Britain and Northern Ireland, 1955-1958.

Professor Brian W. Windeyer was elected to represent the New South Wales and Queensland Branches on the Central Council for the unexpired portion of the late Dr. Isaac Jones's term of office, i.e. until the Annual Representative Meeting in 1958.

Articles of Association.

At an extraordinary general meeting of the Association held on Thursday, December 12, 1957, Articles 37, 40 (1), 53 and 56 were amended. These articles provide for the election of vice-presidents.

As a result of the amendments, the Articles revert to those which were in existence prior to February 27, 1953.

Annual Branch Prize for an Essay on a Scientific Subject.

The subject for the Annual Branch Prize for the year 1957 was "The Acute Abdomen". Only one entry was submitted, but the judges considered it to be worthy of the award. The author of the essay was Dr. J. B. Binks, Griffith, New South Wales.

Dr. W. Fenwick, M.C.

In 1955 the Annual Representative Meeting of the British Medical Association decided to institute a Book of Valour in which should be recorded by decision of the Council of the Parent Body heroic deeds performed by medical practitioners.

On August 27, 1956, a young boy fell down a 150-feet mineshaft at Grenfell, N.S.W.

Dr. W. Fenwick, who had been in practice in the town for many years, twice descended on a rope in an attempt to rescue the boy, but foul air necessitated him being hauled to the surface. The boy was subsequently reached by a third person in a bosun's chair, equipped with a gas mask.

The attention of the Branch Council was drawn to the matter in a minute received from the Mayor of Grenfell. A copy of the minute was forwarded to the Council of the Parent Body with a recommendation that the name of Dr. Fenwick be included in the Book of Valour. A reply was received from the Parent Body advising that it had decided to record the deed in the Book of Valour.

Dr. W. Fenwick is the first Australasian practitioner to have this honour conferred upon him.

National Health Service.

"Health of a Nation."

On October 21, 1957, a special Newsletter was sent to all members giving a précis of a report of a joint committee of the Australian Labour Party, New South Wales Branch, and the New South Wales Labour Council.

The joint report expressed the opinion that there should be introduced and maintained by the Commonwealth Government a complete health service free to all, based on the British National Health Service.

Public Relations.

During the year the Council engaged the services of a leading firm of public relation experts.

The Association sponsored a supplement "Handbook to Health" which was published by *The Women's Weekly* in its issue of March 12, 1958. This is part of a wide television, radio and Press programme which aims at educating the public in medical matters. *A Monthly Newsletter* is also being sent to the editors of the major country newspapers. More than 80 editors have published the newsletters in their papers.

Lunacy Act.

In the latter part of 1956 advice was received by Council that a special committee had been set up by the Minister for Health to advise on amendments to the *Lunacy Act*. This Committee extended an invitation to the Association to express its views on the matter. After referring the matter to the Section of Neurology, Psychiatry and Neurosurgery, the Council appointed a special committee consisting of two psychiatrists and a general practitioner to consider the matter.

This special committee drew up a number of recommendations which were adopted by Council and forwarded to the committee appointed by the Minister for Health.

The recommendations were as follows:

1. That the present Act needs completely redrafting in terms of knowledge of mental illnesses and treatment gained since the original Act and its amendments. To simplify the Act, many provisions in the present Act would be dealt with by regulation by an authority set up under the Act.

2. That there is need for a revision in terminology. Such terms as "Lunacy" and "Reception House" should be replaced with "Mental" and "Observation Hospital or Ward", "Asylum" and "Hospital for the Insane" with "Mental Hospital", "Master in Lunacy" with "Master in Equity" and

"Lunacy Court" with "Equity Court". It is further recommended that the term "Observation" should not exclude treatment and that the word "Idiot" be deleted from the Act.

Generally speaking, patients should be classified into two groups:

(*a*) Mentally ill patients (including mental infirmity of old age).

(*b*) Patients of subnormal personality (incapable of leading an independent life).

Special consideration should be given to the classification of patients with psychopathic personalities.

3. That control of mental hospitals should remain vested in the Inspector-General and the Deputy Inspector-General.

4. That increased suitable accommodation in mental hospitals should be made available for voluntary admissions. The conditions of such admissions should be similar, re administration, to admissions to a general hospital. The present conditions of discharge of a voluntary patient from a mental hospital should be retained.

5. That suitable observation wards should be established in general hospitals, both in the city and country.

6. That the law should be amended to simplify the procedure whereby a patient, the subject of a Schedule 2A, is removed from a private residence or property to an observation ward. Police or, preferably, suitably trained lay officers should be given power to enter properties and effect the removal of patients to mental hospitals.

7. That the form of Schedule 2A should include provision for the attending practitioner to make a written support for the Schedule 2A and that treatment should be made available to a patient admitted to an observation hospital on such a Schedule 2A.

8. That legal formality should be at a minimum and facilities should be available for patients admitted on a Schedule 2A to an observation hospital to receive treatment for a period of three to six months without further formality unless it is necessary because of the patient's objection to resort to certification.

9. That senile or aged people should be dealt with under a different section, again reducing formality to a minimum but with adequate care of property. Such patients should be certified as being unable to control and administer their property and affairs but *should not be certified as insane*. Special units should be authorized for their care.

10. That more enlightened legislation is desirable for the treatment and detention of mental defectives.

11. That actual certification proceedings should not be dealt with in open court as at present, though legal representation should be available if required, that there should be simplification of such court formality and that preferably the machinery of the Act dealing with admissions should not be in the hands of the police as at present.

12. For the purpose of the Act a psychiatrist should be defined as a legally qualified medical practitioner having special experience in psychiatry.

13. That local authorities should be appointed to control local facilities wherever they exist or can be established to treat mental illness.

14. That there is a desirability to establish community care, residential service and rehabilitation for mentally ill patients.

Committee on Medical Education.

At an extraordinary general meeting of the Association held at 8 p.m., Wednesday, February 12, 1958, in the Robert H. Todd Assembly Hall, the following resolutions were adopted:

1. "That the N.S.W. Branch of the British Medical Association should immediately form a committee to discuss medical education in New South Wales with special reference to the Murray Report."

2. "The committee be constituted as follows, viz.:

i. One member nominated by the active teaching staff of each of the four teaching hospitals of Sydney.

ii. Three representatives nominated by the Council of the New South Wales Branch of the British Medical Association.

iii. A representative nominated by the active teaching staff of each of the three special teaching hospitals.

iv. A representative nominated by each of the following Colleges: Royal Australasian College of Physicians, Royal Australasian College of Surgeons, Royal College of Obstetricians and Gynæcologists and the College of General Practitioners.

v. The Senate of the University of Sydney be invited to nominate:

(*a*) one representative of the Senate,

(*b*) four (4) representatives of the teaching staff of the Faculty of Medicine of the University of Sydney.

vi. The Council of the New South Wales University of Technology be invited to appoint a representative.

vii. The Post-Graduate Committee in Medicine be invited to appoint a representative on the committee.

viii. The committee be granted power to coopt."

3. "The committee to make its report urgently to the Council of the New South Wales Branch of the British Medical Association and for Council to take such action as it deems necessary."

Unfortunately the University of Sydney was unable to accept the invitation to appoint a representative and up to the time of preparation of this report the University of Technology had not made a decision.

It is the intention of the committee to consider all aspects of medical education, undergraduate, graduate and post-graduate. As medical education covers a vast field it may be some time before the committee completes its full report, but interim reports will be published from time to time.

Medical Practitioners' Act.

Registration of Alien Practitioners.

In the early part of 1957 amendments were made to the *Medical Practitioners' Act* providing for the registration of alien practitioners.

Under Subsection 3 of Section 21c of the Act, a person subject to fulfilling certain conditions, may be licensed to practise under supervision as a medical officer in a State hospital, mental hospital, public hospital, private hospital or any other institution as the Minister may direct. After having completed to the satisfaction of the Minister a period of twelve months' service in the aforementioned capacity, the practitioner may be allowed to practise without supervision as a medical officer in a State hospital, mental hospital, public hospital, private hospital or other institution or as an assistant to such registered person as the Minister may direct, subject to certain conditions, one of which is that he must pass a prescribed test. It is possible that such person may fail in this test but yet under a further amendment to the Act (Subsection 8A of Section 21A) the Minister would be entitled to issue him a certificate of "regional registration" without examination, if the Medical Board decided there were otherwise no suitable applicants. Prior to the amendment the Medical Board had the right to require an applicant for regional registration to undergo an examination (not being a written examination). This right was withdrawn as the result of the amendment.

A strong protest against the amendment to the Act was made to His Excellency the Governor of New South Wales. In making this protest it was stated:

"There are thus two vital principles involved in these amendments, the first of these the registration of practitioners without some adequate examination, and the second, the power of the Minister to make an appointment for regional registration of a person who may be entitled to apply for a licence in terms of Subsection 4 of Section 21c.

"The decision of the Parliament to provide for the registration of persons without examination may well lead to a deterioration in the present high standard of medical practice of which this State has good cause to be proud.

"The power of the Minister to appoint for regional registration a person who may fail in an examination is certainly not in the public interest."

Medical Register.

The *Medical Practitioners' Act* was also amended to provide for the printing of a list of the names and addresses of medical practitioners entered in the register. Previously copies of the register were printed giving not only the names and addresses of practitioners but the registration number, the date of registration and the degrees of each practitioner. A similar amendment was made to the *Dentists' Act*, the *Opticians' Act*, the *Physiotherapists' Registration Act* and the *Pharmacy Act*.

The New South Wales Medical Board was informed that the Council was of the opinion that the new register was practically useless to persons and bodies requiring information about doctors, was a disgrace to the State of New South Wales and should be remedied immediately. The amending of the Act so as to provide for the new type of

register was defended by the Under Secretary for Health on the grounds of economy and promptitude in publication of the list of doctors and addresses.

The New South Wales Branch of the Australian Dental Association decided to join with the Association in a joint approach to the Minister to have the Acts amended again to provide for a return to the previous type of register.

Hospital Policy.

Staffing of Country Hospitals.

The following policy in regard to the staffing of public hospitals was adopted during the year:

i. The basic policy should be one of visiting staffs as against full-time staffs.

ii. Base hospitals should be staffed by classified staffs.

iii. In hospitals other than base hospitals staffing should be on a general practitioner basis.

iv. In the case of hospitals other than base hospitals, as specialists become available or general practitioners with higher qualifications become available in the district they should be given the opportunity of being appointed to the hospitals as members of a classified staff.

v. Whenever possible the services of consultants should be made available to hospital staffs.

Salaries of Specialists in Hospitals.

Following consultations with representatives of the general teaching hospitals in 1956, submissions were made to the Hospitals Commission concerning the salaries which should be paid to specialists holding full-time appointments in public hospitals. Finally the Hospitals Commission forwarded a determination it had made in which, briefly, the salaries of full-time specialists in hospitals are:

1. Specialist—a practitioner who has served two years in a general medical post in an approved hospital, not less than three years in trainee specialist training in an approved hospital or institution and has obtained a higher medical qualification appropriate to the specialist:
   (*a*) all teaching hospitals, Royal Newcastle Hospital and Prince Henry Hospital, £2,700 × £200 to £3,500 *per annum*;
   (*b*) other hospitals approved by the Hospitals Commission to have specialists (and/or junior specialists), £2,500 × £100 to £3,000 *per annum*.
2. Junior specialist—a practitioner who has completed the five years referred to above but has not obtained his higher qualification or who has met all of the requirements of a specialist but is considered by the hospital to need further experience as a junior specialist, £2,000 × £100 to £2,300 *per annum*.
3. Directors of services—where in the opinion of any of the hospitals mentioned in 1 (*a*) above, any specialist employed by it is required to undertake extensive administrative duties, it may pay that employee such additional remuneration as may be approved by the Hospitals Commission up to a maximum of £200 *per annum*.

Placement of Resident Medical Officers.

Some years ago there were more successful candidates of the final degree examination in Medicine, University of Sydney, than there were vacancies for resident medical officers in the State. As a result, a number of the successful candidates had to be sent to other States and even to New Zealand. Then followed a decrease in the numbers undertaking the medical course at the University, with a consequent decrease in the number of graduates. As a result of this decrease, hospitals which had taken resident medical officers whilst the numbers were large have been unable to obtain them following recent examinations.

Over the years the Committee for Placement of Resident Medical Officers has made its allocations to hospitals, other than the teaching hospitals, on the basis of the successful candidate's wishes and his place in the order of merit list issued by the University of Sydney. This has resulted in the teaching hospitals and the metropolitan district hospitals taking virtually all of the successful candidates of the examination.

The Council considered the matter to be one of great importance, and after a good deal of consideration it made the following recommendations:

(*a*) The first year of appointment as a resident medical officer be regarded as part of the training of the doctor.

(*b*) The training of recent graduates can be carried out adequately only in (i) teaching hospitals, or (ii) hospitals with consultants who are also attached to the staffs of teaching hospitals, or (iii) other hospitals which are considered to be of a standard equivalent to those mentioned in sub-clauses (*b*) (i) and (*b*) (ii).

(*c*) If and when the supply of recent graduates exceeds the number of appointments available in such aforementioned hospitals approved for training, then, but only then, should recent graduates be appointed as resident medical officers to other hospitals and such appointments should be made subject to the proviso that the hospital employs a senior resident medical officer.

(*d*) After the first year of hospital training, resident medical officers who have not received second year appointments at their hospitals should be encouraged to go to the larger country hospitals for practical experience.

(*e*) The teaching hospitals be encouraged to reengage such resident medical officers as have spent a second year in country hospitals gaining experience.

Operating Theatres—Handling of Swabs, Sponges, and Instruments, etc.

Following reference of the matter to the Section of Surgery, the following recommendations were made by Council with a view to standardizing the technique in all operating theatres in connexion with the handling of swabs, sponges, and instruments, etc.

1. All instruments, sponges and throwaways should be checked by two persons at the following times: (*a*) when packaging for sterilization; (*b*) in the theatre prior to the operation; (*c*) on closing the wound; (*d*) on cleaning up the theatre.
2. Radio-opaque sponges and throwaways only should be used in the theatres.
3. A uniform number of sponges should be put up in each package. It is suggested that packages of five and ten only to be used.
4. Counting racks or similar devices for quick checking would assist in the theatre.
5. Throwaways should be coloured and reserved as follows: *white* for use of the surgeon; *green* for use of the anæsthetist; *blue* for use anywhere else in the hospital away from the theatre.

Nurses' Registration Board.

The Nurses' Registration Board provides for the constitution of a Nurses' Registration Board which shall consist of fourteen (14) members.

Of these fourteen members, four (4) are medical practitioners, one being the Director-General of Public Health, one the Inspector-General of Mental Hospitals, one a teaching professor in the School of Medicine in the University of Sydney nominated by the Senate of the said University, and one a legally qualified medical practitioner nominated by the Post-Graduate Committee in Medicine.

As the medical profession as a whole is intimately associated with the working of the nursing profession and in fact is instrumental in training nurses, it was felt by Council that the profession as a whole as represented by the New South Wales Branch of the British Medical Association should be represented on the Nurses' Registration Board. Accordingly it made representations to the Minister for Health that the *Nurses' Registration Act* should be amended to provide for the representation of the New South Wales Branch of the British Medical Association on the Board.

In his reply the Minister assured the Council that the association of the medical profession with the training and examination of nurses in New South Wales is thoroughly appreciated, but as the Nurses' Registration Board at present includes four medical practitioners, the composition of the Board is considered to serve adequately the purposes for which it was established.

Salk Vaccine.

During the year the Council advised Federal Council that it was of the opinion that a request should be made of the Commonwealth Government to make available supplies of Salk vaccine for use in private practice when it became practicable to do so.

Subsequently a letter was received from the Federal Council advising that it had taken the matter up with the Minister for Health. In the course of his reply to the Federal Council the Minister gave an assurance that the vaccine would be made available to the general practitioner as soon as conveniently possible.

Asian 'Flu Epidemic.

Appointment of an Advisory Committee by the Minister for Health.

During the Asian 'flu epidemic of last year the Director-General of Public Health advised that the Minister for Health had decided to establish an Advisory Committee in connexion with the influenza epidemic.

Following a request made by the Director-General, the Council submitted the following names as representatives of the Branch on the committee, viz.: Dr. G. L. Howe, Dr. E. F. Thomson, Dr. D. G. Hamilton, Professor F. M. de Burgh, and Miss Phyllis Rountree, D.Sc.

Workers' Compensation Act, 1926-1957.

Negotiations have been proceeding with the licensed insurers for some time in connexion with the following matters, viz.:

(*a*) the introduction of a new schedule of fees to replace Schedule H,
(*b*) the introduction of a schedule for specialist general surgeons,
(*c*) fees for X-rays by general practitioners,
(*d*) establishment of clinics for the treatment of injured workers.

Finality has not yet been reached in these matters.

Medical Certification.

During the year communications were addressed to various employer organizations pointing out that it was common practice in industry for an employee claiming sick pay for an alleged illness when he was not attended by a medical practitioner to be told by the employer or his representative to "go and get a medical certificate". It was further stated in the communication that the doctor was not in a position to supply evidence of the patient's illness at the time and refusal on his part to issue a certificate often engendered ill feeling. In the interests of all parties the Council recommended to the employer organizations that they ask their members not to advise employees to endeavour to obtain medical certificates in support of their claims for sick pay unless the employee was actually under medical care during his illness. The cooperation of the Employers' Federation of New South Wales and the Metal Trades Employers' Federation was readily forthcoming.

To help in the matter, a standard form of medical certificate was prepared by Council. A copy of this certificate was sent to all members with a recommendation for its use.

Annual Ball.

The second annual ball, which proved to be a great success, was held at the Trocadero on Wednesday, July 24, 1957. The guest of honour was Dr. H. C. Colville, President of the Federal Council. Mrs. Colville was unfortunately unable to attend owing to her absence abroad.

The next ball will be held at the Trocadero on Wednesday, July 23, 1958.

Golf Tournament.

The annual golf tournament for the British Medical Association Cup, presented by the late Dr. H. C. Rutherford Darling, was held on the golf course of the Australian Golf Club at Kensington on Tuesday, November 19, 1957.

The winner was Dr. J. C. Fulton, and the runner-up, Dr. R. E. H. Holliday.

The British Medical Agency of New South Wales Limited.

The annual meeting of The British Medical Agency of New South Wales Limited was held on October 1, 1957, the annual report being presented by the Chairman, Dr. George Bell.

The Agency's activities for the year resulted in a small surplus for the year being somewhat less than the profit for the preceding year.

NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

Balance Sheet as at December 31, 1957.

	£ s. d.	£ s. d.
FIXED LIABILITIES.		
Debentures—		
51—4% Series "A" at £10 each	510 0 0	
249—4·65% Series "B" at £50 each	12,450 0 0	
197—4·65% Series "C" at £10 each	1,970 0 0	
	14,930 0 0	
General Reserve Fund (used in the Branch Business)	2,272 0 0	
		17,202 0 0
CURRENT LIABILITIES.		
Sundry Creditors	2,821 16 9	
Deposit at Call	700 0 0	
Commercial Banking Co. of Sydney Limited—Branch and Premises Current Accounts	10,603 9 10	
		14,125 6 7
OTHER CREDIT BALANCES.		
Subscriptions Paid in Advance—Suspense Account	87 15 8	
Provision for Taxation	4,676 17 0	
Provision for Long Service Leave	2,000 0 0	
Provision for Painting Building	6,400 0 0	
Provision for Handbook and Library Catalogue	1,750 0 0	
Superannuation Fund Reserve Account, as *per contra*	2,929 17 10	
		17,844 10 6
Accumulated Funds—		
Balance as at December 31, 1956	110,479 17 1	
Add Surplus for Year—Branch	4,387 17 6	
Premises	943 16 7	
		115,811 11 2
		£164,983 8 3

	£ s. d.	£ s. d.
FIXED ASSETS.		
Land and Buildings—B.M.A. House, at Cost *less* Depreciation	123,512 9 0	
Library, at Cost *less* Depreciation	8,963 4 0	
Office Furniture and Equipment, at Cost *less* Depreciation	3,141 8 11	
Debentures in Other Companies—Australasian Medical Publishing Co. Ltd.—Face Value	15,400 0 0	
Deposit on Debentures	91 0 1	
Commonwealth Treasury Bonds—Face Value	6,000 0 0	
		157,108 2 0
CURRENT ASSETS.		
Sundry Debtors (after making provision for Doubtful Debts, Sundry Tenants, Rents, etc.)	3,169 5 3	
Cash on Hand	21 10 8	
		3,190 15 11
OTHER DEBIT BALANCES.		
Prepaid Insurances, Rates, etc.	1,754 12 6	
Superannuation Fund Investment Account, as *per contra*	2,929 17 10	
		4,684 10 4
		£164,983 8 3

Sydney,
February 17, 1958.

G. L. HOWE, President.
W. F. SIMMONS, Honorary Treasurer.
J. F. WALKER, Accountant.

We have examined the foregoing Balance Sheet with the Books of Account of the New South Wales Branch of the British Medical Association, and having obtained all the information and explanations we have required, we are of the opinion that such Balance Sheet is properly drawn up so as to exhibit a true and correct view of the state of the Company's affairs according to the best of our information and the explanations given to us and as shown by the Books of the Company. In our opinion, the Register of Members and other records which the Company is required to keep by the New South Wales Companies Act, 1936, or by its Articles have been properly kept.

F. W. DUESBURY & Co.,
Chartered Accountants (Aust.).

During the financial year a large number of introductory letters were issued to doctors enabling them to obtain prompt delivery of Holden cars. This service was particularly helpful in the early part of the year, when there was otherwise a long delay in the delivery of these cars.

The Directors are confident that an increasing number of the medical profession will avail themselves of the Agency's services, which are provided for their assistance and convenience.

Mr. C. R. Strange, who had been manager of the Agency since February, 1945, retired on December 31, 1957. In their report the directors stated they wished to place on record their appreciation of his services during this period. Mr. P. R. E. Murnin has been appointed to succeed Mr. Strange.

Medical Finance Limited.

The annual meeting of Medical Finance Limited was held on October 1, 1957.

The report which was presented by the Chairman, Dr. George Bell, showed a small increase in profit on the previous year. This increase was the result of a higher interest received during the year from the larger amount on loan accounts. Several loans were repaid towards the end of the financial year, this placing the company in a position to consider applications for assistance.

Premises Revenue Account.

The Premises Revenue Account discloses a net surplus of £944 for the year ended December 31, 1957, as against a net deficit of £144 for the year ended December 31, 1956, thus showing an increase of £1,088 in net revenue earned. This increase is accounted for by a net increase in income of £3,935, less a net increase of expenditure of £2,847 as detailed in accompanying comparative statement.

A comparison of percentages of expenditure to rent revenue with those of December 31, 1956, is as follows:

	1956.	1957.
Percentages of Expenses to Revenue ..	100·48	97·2
Percentages of Surplus to Revenue ..	·48	2·8
	100%	100%

The percentage of rent revenue, expenses and depreciation and the percentage of net surplus for the year to the capital value of the land and building (British Medical Association House) is shown by the books at December 31, 1957, namely £123,512, with the previous year's percentages in parentheses, are as follows:

Rent Revenue (including amount charged for British Medical Association Branch Offices, etc.)		26·9%	(23·18%)
Sundry Expenses, Interest, Provision for Painting, etc.	23·87%		(21·09%)
Depreciation of Building ..	2·27%	26·14%	(2·22%)
		·76%	(·6%)

Financial Statement.

The Council has pleasure in presenting to members the balance sheet and accounts in respect of the financial year which terminated on December 31, 1957. The net surplus of revenue over expenditure amounted to £5,332 after making provision for all known expenditure.

The sum of £4,875 has been written off for depreciation of the building (British Medical Association House), plant, office furniture, equipment and the library.

The sum of £800 has been provided out of the current year's revenue to create a reserve for painting of the exterior of the building, £150 for long service leave, £750 for a library catalogue and £1000 for a "Handbook for Qualified Medical Practitioners".

G. L. HOWE,
President.

The balance sheet of the Branch and the income and expenditure account of the Branch and of the premises were received and adopted on the motion of Dr. W. F. Simmons, seconded by Dr. A. J. Murray.

ELECTION OF OFFICE-BEARERS.

Dr. G. L. Howe then announced that the following had been elected to the Council as representatives of the general body of members for the ensuing year: Dr. M. S. Alexander, Dr. E. A. Booth, Dr. D. A. Brown, Dr. J. F. C. C. Cobley, Dr. D. G. Hamilton, Dr. P. J. Kenny, Dr. R. H. Macdonald, Dr A. J. Murray, Dr. T. Y. Nelson, Dr. K. C. T. Rawle, Dr. W. F. Simmons, Dr. R. J. J. Speight, Dr. E. S. Stuckey, Dr. E. F. Thomson, Dr. P. A. Tomlinson, Dr. D. A. Warden.

NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

BRANCH ACCOUNT.

Income and Expenditure Account for the Year Ended December 31, 1957.

	£ s. d.	£ s. d.
To Salaries	14,114 12 0	
„ Rent—Offices, etc.	1,200 0 0	
„ Printing and Stationery	2,019 1 4	
„ Stamps and Telegrams	1,085 16 1	
„ Telephones	346 12 8	
„ Travelling Expenses—General ..	817 9 11	
„ Code Address	3 8 0	
„ Insurance	57 14 8	
„ Exchange and Bank Charges ..	15 19 0	
„ Refreshments—Meetings	80 16 0	
„ Newspapers and Advertising ..	49 9 10	
„ Sundry Petty Expenses	63 14 7	
„ Tea Money	128 6 11	
„ Federal Council	4,910 0 6	
„ Legal Expenses	124 19 0	
„ Repairs and Maintenance—Equipment	120 16 6	
„ Payroll Tax	244 7 0	
„ Medical Benefits Fund—Staff ..	23 4 3	
„ Staff Superannuation Fund ..	967 10 7	
„ Incidental, Travelling and Entertainment Expenses	324 0 0	
„ Bank Interest	142 5 9	
„ Stamp Duty	68 11 6	
„ Laundry	28 6 7	
„ Branch Prize	100 0 0	
		27,037 2 8
„ Allowance for Depreciation of—		
Library	1,859 16 5	
Furniture and Equipment ..	166 8 0	
Long Service Leave	100 0 0	
		2,126 4 5
„ Provision for—		
Handbook	1,000 0 0	
Library Catalogue	750 0 0	
		1,750 0 0
„ Surplus for Year transferred to Accumulated Funds		4,387 17 6
		£35,301 4 7

	£ s. d.	£ s. d.
By Subscription Revenue		41,954 3 6
Less Proportion due to—		
British Medical Association	6,246 13 6	
MEDICAL JOURNAL OF AUSTRALIA	1,937 5 0	
		8,183 18 6
		33,770 5 0
„ Interest—		
Australasian Medical Publishing Co. Ltd.—Debentures ..	847 13 1	
Commonwealth Treasury Loans	178 15 0	
Fixed Deposit	28 2 6	
		1,054 10 7
„ Rent—Assembly Hall	386 8 0	
„ Broadcasting and Journalist Fees	36 18 0	
„ Accountancy Fees	53 3 0	
		476 9 0
		£35,301 4 7

Elected as representing women members: Dr. Mary C. Puckey.

Elected as representing the Public (Government) Medical Services: Dr. L. W. Wing.

Elected as representing country local associations: Dr. B. A. Cook, Dr. B. W. Monahan.

Elected as representing metropolitan local associations: Dr. K. S. Jones, Dr. C. R. M. Laverty.

Messrs. F. W. Duesbury and Company were elected auditors for the ensuing year.

ELECTION OF REPRESENTATIVES OF THE BRANCH AT THE ANNUAL REPRESENTATIVE MEETING OF THE BRITISH MEDICAL ASSOCIATION, 1958, BIRMINGHAM.

On the motion of Dr. E. F. Thomson, seconded by Dr. P. A. Tomlinson, it was resolved that the appointment of representatives of the Branch to attend the annual meeting of the British Medical Association should be left in the hands of the Council.

PRESENTATION OF THE ANNUAL BRANCH PRIZE FOR AN ESSAY ON A SCIENTIFIC SUBJECT.

Dr. G. L. Howe announced that the annual Branch prize for an essay on a scientific subject had been awarded for the second time. The subject set for the 1957 essay was "The Acute Abdomen", and the winner was Dr. J. B. Binks. Dr. Howe said that it was interesting that Dr. Binks was a country practitioner, and all present were glad that he was able to attend the meeting to receive his prize.

INCOMING PRESIDENT'S ADDRESS.

Dr. A. W. Morrow delivered his incoming president's address on the subject of "The Association, the Colleges and the Future" (see page 621).

A vote of thanks to Dr. Morrow for his address was carried on the motion of Dr. M. S. Alexander, seconded by Dr. L. W. Wing.

INDUCTION OF PRESIDENT.

Dr. G. L. Howe inducted the President for the year 1958-1959, Dr. A. W. Morrow, and invested him with the presidential badge of office. Dr. Morrow thanked the members for his election.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

MEDICAL POPULATION OF AUSTRALASIA.

[From the *Australasian Medical Gazette*, May, 1893.]

THE total number of registered practitioners recorded in Australasia, excluding Fiji and other South Sea Islands, is 2410 (one to every 1660 persons), of whom 691 reside in New South Wales (one to 1708), 204 in Queensland (one to 2304), 181 in South Australia (one to 1796), 812 in Victoria (one to 1441), and 40 in Western Australia (one to 1375). A total of 1928 on the Australian Continent (one to 1631). In Tasmania there are 93 practitioners (one to 1666) and in New Zealand 389 (one to 1799).

Of these there are 291 or 12·07 per cent. who have graduated at Colonial Universities, viz., 222 at the Medical School of the University of Melbourne (opened in 1862), 47 at that of Sydney (opened in 1883), nine at the one in Adelaide (opened in 1885) and 13 at the University of Otago, N.Z. Those practitioners who have taken colonial degrees *ad eundem gradum* are of course not included. It must be assumed that these 291 colonial graduates are almost entirely natives of these colonies, and as there are quite as many Australians and New Zealanders who have pursued their studies in the old country and taken their degrees at some University in Great Britain, it follows that fully 25 per cent. of all colonial practitioners must be natives of Australia or New Zealand. There are nine ladies amongst the legally qualified medical practitioners—viz., five in Victoria, two in New South Wales, one in Queensland, and one in South Australia, four of them being possessors of Australian degrees.

Correspondence.

STAFFING OF BASE HOSPITALS.

SIR: In his letter of April 7, 1958, published in the issue of THE MEDICAL JOURNAL OF AUSTRALIA of April 26, 1958, Dr. J. G. Glasson expresses his concern at the policy adopted by the Council of the New South Wales Branch in regard to the staffing of country base hospitals.

I can assure Dr. J. G. Glasson that the policy "that base hospitals should be staffed by classified staffs" was adopted by Council only after prolonged and serious consideration. On matters such as this the Council has an open mind and it will be only too pleased to have suggestions from members as to the manner in which the hospital policy may be improved. If Dr. J. G. Glasson is of the opinion that the policy in regard to country base hospitals should be amended, then it is recommended that he refer the matter to his Local Association.

Yours, etc.,
HUGH HUNTER,
Assistant Medical Secretary,
British Medical Association,
New South Wales Branch.

135 Macquarie Street,
Sydney,
April 30, 1958.

ŒDEMA AND SODIUM CHLORIDE.

SIR: Whilst working as a biochemist in a hospital four years ago the undersigned became increasingly alarmed at the rapid onset of œdema in her own case; hence it is considered worth recording what subsequently transpired with respect to the intake of sodium chloride. It must be stressed that the situation was a puzzling one; the undersigned was becoming daily more and more breathless upon exertion, with flushed face, and it was a painful matter to remove shoes from œdematous feet. There did not seem to be any underlying abnormality of cardiac function etc. It was only after giving the matter considerable thought that it was decided that the diet could perhaps be lacking in an adequate intake of one essential item, namely sodium chloride. It was therefore by way of an experiment that sodium chloride was consumed in quite large quantities (approximately one dessertspoonful A.R. grade per cup of water two to three times per day) fortified by an experience of the considerable benefit derived from doing the same thing on a smaller scale in the tropics. Fluid was excreted in positively astonishing quantities (via the kidneys), so much so that the writer then began to wonder if she might prove to be a case of *diabetes insipidus*. This did not prove to be the case, however. It is of further interest that at the time of sodium chloride deficiency the characteristic taste associated with this electrolyte was at first almost absent.

(MISS) L. E. A. WRIGHT, M.Sc.

The Sydney University Women's Union,
Manning House,
The University of Sydney.
April 11, 1958.

EPISIOTOMY.

SIR: The need for clinical clarity prompts this further statement on episiotomy. Slavish preservation of an "intact" perineum and the routine use of ether during delivery belong to the past, not because of fashion, but because safer and more efficient methods have replaced them. Episiotomy has for long been established as an important everyday procedure in obstetrics. Apart from its clear place in complicated delivery it is also generally desirable in the *primigravida* with an uncomplicated labour. Without an adequate episiotomy and careful repair the majority of *primigravidæ* cannot regain their full vaginal tone, contractility and sensibility. That is why so many women date diminution or loss of full physical satisfaction in sexual life from their first confinement. Failure to perform an efficient episiotomy is also a common cause of low rectocele, cystocele and stress incontinence, but not of uterine prolapse, for the uterine supports are at a much higher level than the perineum and lower vagina. *Primiparæ* who have had an adequate episiotomy will usually require smaller episiotomies at subsequent deliveries and, at times, no episiotomy.

Today there are many methods available for the relief of pain during delivery, the safest and most satisfactory of which include local infiltration with "Xylocaine", pudendal block, caudal block, and inhalation analgesia with trilene or nitrous oxide and oxygen. A general recommendation for the use of ether, the dangers of which have been amply demonstrated and publicized, is therefore not warranted.

Yours, etc.,

111 Collins Street,
Melbourne, C.1,
April 21, 1958.

A. M. HILL,

NEPHRITIS IN QUEENSLAND.

SIR: Dr. David Henderson's *magnum opus* on this topic (M. J. AUSTRALIA, March 22, 1958) is commendable and is one of the few writings giving due credit to A. J. Turner and Lockhart Gibson. The late E. S. Meyers knew these men well and was often heard to say that they had not (then) received the recognition they merited. Turner, in his Jackson Lecture of 1938, referred to his paper of 1892 on lead poisoning in Queensland, refraining from discussing again the clinical details, saying "there is no need to fight this battle o'er again and thrice to slay the slain". He went on to say: "Gibson gave us the key to this riddle (of the source of the lead). He showed us that the paint inside the house, once it is dry, is harmless; and that outside weathering of painted veranda rails, fences, and gates was the danger. Most illuminating was the remark of one of the mothers, whom he was questioning: 'And this is the only one of my children who bites his nails'."[1] The justification of these resurrections is, I trust, seen in the points already made.

It is fitting that Jefferis Turner should have entitled his Jackson Lecture "Experiences in Preventive Medicine", and fitting, too, that a distinguished Queensland graduate should have followed the subject so far and so thoroughly towards its conclusion.

Yours, etc.,

Sydney,
April 18, 1958.

R. I. MEYERS.

MEDICAL EDUCATION.

SIR: I have read Dr. Addison's letter (M. J. AUSTRALIA, April 26, 1958) with interest. A compulsory six-month trainee nursing period is in force in Germany since about 1939. This can be done either before the preclinical studies or during the holidays of the first two years. The superintendent of the hospital recognized for the purpose must furnish a confidential report about his impression of the trainee's character and aptitude for the profession, which the university takes into account. Fit males received further 12 to 18 months' ambulance training with the armed forces.

As far as my own experience with the method goes, it seems to be important that the trainee nurse is rotated and not left in one place (say in the theatre section) all the time. The method proved extremely valuable in the great national emergencies and helped me a lot as superintendent of a medium-sized hospital. It also helps in general practice (e.g., in first aid, nursing advice and first aid teaching). It appeared to me not so important for specialist practice.

The greatest curriculum deficiency which struck me both in Germany as well as in Australia, was the virtual absence of teaching in general practice at the undergraduate level, and the chanciness of its later acquisition. The answer here seems to me to lie not in "apprenticeships", but in a university chair and department of general practice, based on "Teaching Practices". These could be staffed by salaried full-time general practitioner teachers and research workers, who, apart from the time needed for teaching and research, would be obliged to make a profit comparable to that of a private practice. This profit should be available for research. In this way the practice may keep its feet on the ground. Several types of teaching practices could operate under the coordinating supervision of the professor: the one-man country practice, the partnership practice, and the city practice.

When the formation of a new medical school is contemplated, the need to put general practice on an academic basis should be a primary consideration. Then it would not be necessary for each graduate to acquire haphazardly his individual experiences in this field, a common nomenclature for the everyday syndromes, which form the greatest economic burden to national health, could be evolved. Morbidity studies would not be limited to the heroic efforts of a few idealists, and improved domiciliary techniques could be developed, among many other things. Economies could be intelligently studied. An optimum doctor/population ratio might be determined and training adjusted accordingly. The public in general would benefit more than from improvements in any other field.

Yours, etc.,

Tea Gardens,
New South Wales,
April 27, 1958.

HANNS PACY.

[1] M. J. AUSTRALIA, 1938, 2: 805.

Obituary.

PHYLLIS MARGERY ANDERSON.

PHYLLIS MARGERY ANDERSON, who died suddenly at her home in Sydney on November 28, 1957, left a deep and abiding impression. Her personal qualities and her scientific and other contributions to medicine influenced and enriched individual lives, her own profession and the community.

Educated at the Methodist Ladies' College, Burwood, and the University of Sydney, Phyllis Anderson graduated in medicine in 1925. Her medical career had two main phases, the first in the Department of Pathology at the Royal Alexandra Hospital for Children, the second on the teaching staff of the Department of Bacteriology in the University of Sydney, where she was first a teaching fellow and later a part-time lecturer. Many qualities contributed to the high standard of her work—a first-class intellect, scientific integrity and fierce personal honesty, human understanding, humility and a powerful sense of humour. For years she was a valued friend of this Journal, reviewing books and preparing abstracts in matters bacteriological and immunological. On one occasion the Editor quoted her opinion anonymously in a leading article and referred to her as "a distinguished Australian pathologist". The phrase quickly became contracted into a nickname, and for many years she was referred to and personally addressed in the office of the Journal as "D.A.P.".

Phyllis Anderson also made a worthwhile contribution to the wider life of her profession. She was particularly prominent among medical women, by whom she was deeply respected and sincerely liked. She was among those present when in 1928 a gathering of interested women created and named the Medical Women's Society of New South Wales. In 1931, when she had returned from a trip abroad, she addressed the Society on "Recent Biochemical Research". She was Secretary from 1935 to 1937, Vice-President in 1938, Treasurer in 1943 and President in 1945-1946. As President she addressed the Society on "The Progress of the Medical Women's Society of New South Wales". She was again Vice-President from 1947 to 1949. From 1950 to the time of her death she represented medical women on the Standing Committee of Convocation of the University of Sydney. From March, 1951, to March, 1954, she served on the Council of the New South Wales Branch of the British Medical Association as representative of the women members of the Association. Her contribution here was valuable and acceptable. Whenever she spoke at a Council meeting, which was not often, she always had something worth while to say, and her remarks were listened to with respect.

A special feature of her life was her interest in music and ballet dancing. She devoted much time to these interests, and in ballet dancing participated in the development of training for ballet in Australia. She was a member of the overseas advisory committee of the Royal Academy of Dancing. Through her advice the committee adopted the ruling that the winner of the scholarship for continuance of the study of ballet dancing overseas must be examined by an orthopædic surgeon and be regarded as "orthopædically" fit to undertake this further training before being officially granted the scholarship. She was also a member of the committee which undertook the social entertainment of Dame Margot Fonteyn during her visit to Australia last year.

Reticent and with a strong dislike of displays of feeling, Phyllis Anderson was nevertheless a very human person. She was a friend to many without making a fuss about it. As a result of her close association with medical students she developed a keen interest in their welfare. She always

strongly supported the Medical Women's Benefaction, and was generous in her help, advice and financial contributions in matters concerning medical women. Many people will remember Phyllis Anderson with gratitude and affection.

PROFESSOR HUGH WARD writes: Phyllis Anderson was an idealist. That idealism was never paraded, but it shone through everything she said and did. Indifferent to her own advancement and reputation, she nevertheless earned what, in the last analysis, is the only thing worth having—the respect of her peers and the affection of all those who knew her well. Trained at the Royal Alexandra Hospital for Children by the late Dr. Tidswell, she found her life's work in the laboratory and the classroom. Happy in both occupations, she will be remembered and missed by half a generation of undergraduates, who, in the phraseology of one of them, "had a great deal of time for Dr. Anderson". From that critical source this is praise indeed. Despite the large

classes, she had a remarkable capacity for treating and remembering students as individuals. Nor did her interest in them cease at graduation, and no one was prouder of their successes or more sympathetic with their difficulties. An appeal for her help, if within her compass, was never made in vain; for instance, the late Dr. Mervyn Archdall called on her regularly for reviews and annotations for his Journal. In truth she was the Good Samaritan. Characteristically, in her will she left nearly everything she had to the University of Sydney, for from the day she entered as a medical student to the day she died the University was her Alma Mater.

A colleague who wishes to remain anonymous writes: The death of Dr. Phyllis Anderson should not be allowed to pass without reference to the part she played when on the pathology staff of the Royal Alexandra Hospital for Children. The writer, who has held resident appointments at Sydney and London, feels that the atmosphere of the Royal Alexandra Hospital for Children in those days was unique. The *camaraderie* between the resident and honorary staffs and amongst the honorary staff themselves was an object lesson. Phyllis Anderson was one of those responsible. What a pleasure for the residents and what unique instruction for the students when the late Dr. Edgar Stephen and other senior honoraries would send for Phyllis Anderson on their rounds. An interesting case would be discussed in the ward, and then the group would go down to the pathology department and be shown specimens and listen to the stories of similar cases. This was pathological practice as Phyllis Anderson thought it should be—a marriage of the clinical and academic. A slide was not just a specimen, but something belonging "to that unhappy baby with the Teddy Bear in the second bed on the right". Later, when at the University, demonstrating malarial parasites, she took a delight in knowing the slide was of the blood of a particularly courageous member of the Australian Imperial Force. She was a lesson in humanity and humility for all women graduates. Many physicians and surgeons occupying senior positions today have reason to be grateful to Phyllis Anderson for their coaching for higher degrees. Though she had no family in Australia, she will be missed by friends, teachers, students and patients.

DR. MARY PUCKEY writes: Dr. Phyllis Anderson was one of the best known women graduates of our time, not only among students and members of her own profession, but also among undergraduates as a whole, many of whom will remember her with affection. She was of a quiet retiring disposition, but keenly alert to all that went on around her, especially affairs affecting the welfare of the university undergraduate. She was interested in people; one of her greatest characteristics was her apparently inexhaustible fund of disinterested kindness. She was always ready to help, either materially or with advice. One could always count on the latter being disinterested and given with a logic and clarity rarely encountered. She had vision and faith in the achievements of medical women. Although never a member of the staff of the Rachel Forster Hospital, she took a great interest in its progress and was the first to see its possibilities as a teaching hospital. Phyllis Anderson will be missed by a wide circle of friends in many walks of life.

MR. DAVID NELSON writes: The death of Dr. Phyllis Anderson will be felt deeply by her students both past and present. Many have known and been grateful for her intense interest in teaching medical students. She had an infectious enthusiasm for her work, and the informal atmosphere of the practical classroom gave her full scope for exercising her faculty of arousing the interest of students. In teaching she always emphasized the practical applications of the basic sciences, and her firm but kind hand guided many in the often difficult transition between pre-clinical and clinical years. Those who knew her better knew her as a widely read and thoughtful person whose conversation was pithy, original and informed—and perhaps were lucky enough to learn from her a little of the habit of accuracy of thought and economy of speech. Above all these qualities of mind was an immense fund of kindness, sympathy and wisdom. Many have been glad to draw on this, even years after graduation. Her untimely death has deprived us, and those who had yet to meet her, of a friend as well as of a respected teacher.

DR. M. GRACE JOHNSTON writes: It was with deep regret that I read of the death of Dr. Phyllis Anderson. She was a most inspiring teacher, whose thoroughness and interest in her work, bacteriology, and whose kindness and untiring help to many students, including myself, gave them a background to carry throughout their professional lives. I have always felt that if any can leave the world a better place for their having lived in it, then their lives have truly been good ones. This is so of Phyllis Anderson.

Medical Societies.

THE CARDIAC SOCIETY OF AUSTRALIA AND NEW ZEALAND.

THE annual meeting of the Cardiac Society of Australia and New Zealand will be held in the Stawell Hall of The Royal Australasian College of Physicians, 145 Macquarie Street, Sydney, on Monday and Tuesday, June 2 and 3, 1958. The provisional programme is as follows:

Monday, June 2.—11 a.m.: "Experiences to Date in Open Heart Surgery at the Royal Prince Alfred Hospital"; medical aspects, Dr. J. G. Richards, surgical aspects, Dr. F. H. Mills; discussion to be opened by Dr. Kenneth N. Morris. 2 p.m.: "The University of Padua and its Medical School", Professor Bryer (by invitation). 2.20 p.m.: "Acute Coronary Insufficiency: The Significance of Plasma Transaminase Activity", Dr. A. Goble and Dr. E. N. O'Brien (introduced).

2.50 p.m.: "Estimation of Valvular Incompetence by Dye Studies", Dr. P. Korner (by invitation). 3.30 p.m.: "Heart Disease in Pregnancy", Dr. M. J. Etheridge.

Tuesday, June 3.—9.30 a.m.: "Studies in Fat Metabolism in Coronary Artery Disease Using an Isotopic Technique", Dr. G. V. Hall, Dr. J. B. Hickie and Dr. P. George (introduced). 10 a.m.: "Inherited Hypertension in Rats", Professor F. H. Smirk. 10.20 a.m.: "Vascular Reactivity in Hypertension", Dr. A. Doyle, Dr. J. R. E. Fraser and Dr. R. J. Marshall (introduced). 11 a.m.: "Voluntary Control of the Circulation", Dr. H. M. Whyte. 11.30 a.m.: "The Measurement of the Extracellular Volume Using I^{131}", Dr. J. B. Hickie, Dr. A. Seldon and Dr. P. George (introduced). 11.45 a.m.: "Factors Contributing to the Apex Beat", Dr. E. H. Roche. 2 p.m.: Clinical meeting at St. Vincent's Hospital.

Inquiries should be directed to the Honorary Secretary and Treasurer of the Society, Dr. J. M. Gardiner, at the Alfred Hospital, Commercial Road, Prahran, S.1, Victoria.

Post-Graduate Work.

MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

Lecture by Dr. Paul White.

THE Cardiac Society of Australia and New Zealand, in conjunction with the Melbourne Medical Post-Graduate Committee, has arranged a lecture by Dr. Paul D. White, of Boston, U.S.A., on "The Epidemiology of Heart Disease". This lecture will be delivered at the B.M.A. Hall, 426 Albert Street, East Melbourne, at 8.30 p.m. on Wednesday, May 28, 1958. The lecture has been made possible through the generosity of the R. T. Hall Trust. All members of the profession are invited to attend without fee.

The Royal Australasian College of Physicians.

OFFICE-BEARERS, 1958-1960.

THE Council of The Royal Australasian College of Physicians has elected the following office-bearers for the term of office 1958-1960; they will take office at the annual meeting of the College on June 3, 1958: *President*, Professor John Hayden (Victoria); *Vice-Presidents*, Dr. Keith Fairley (Victoria), Dr. W. W. S. Johnston (Victoria), Professor F. H. Smirk (New Zealand); *Censor-in-Chief*, Dr. K. B. Noad (New South Wales); *Honorary Treasurer*, Dr. Bruce Hall (New South Wales); *Honorary Secretary*, Dr. H. Maynard Rennie (New South Wales).

SIMS COMMONWEALTH TRAVELLING PROFESSOR.

PROFESSOR M. L. ROSENHEIM, C.B.E., M.A. (Cambridge), F.R.C.P., Sims Commonwealth Travelling Professor for 1958, will visit Melbourne from Sunday, May 11, to Tuesday, May 27, 1958, and Brisbane from Tuesday, May 27, to Sunday, June 2, 1958. In Melbourne his programme will include visits to the teaching hospitals on the following days: Royal Melbourne Hospital, May 12, 13 and 14; Alfred Hospital, May 15 and 16; Royal Women's Hospital, May 19; St. Vincent's Hospital, May 20 and 21; Prince Henry's Hospital, May 22 and 23; Royal Children's Hospital, May 26.

Professor Rosenheim will give the following lectures in Melbourne:

Monday, May 12, at 8.15 p.m., "Cystinuria", lecture to Fellows and Members of The Royal Australasian College of Physicians, at the Royal Melbourne Hospital.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED APRIL 19, 1958.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.[2]	Australia.[3]
Acute Rheumatism	3	5(2)	6(1)	..	..	..	1	..	15
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	1	..	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	6(3)	14(11)	..	..	..	..	2	..	22
Diphtheria	..	2(2)	..	..	..	..	..	..	2
Dysentery (Bacillary)	..	2(2)	4(3)	1(1)	5(3)	..	2	..	14
Encephalitis	..	1	..	1(1)	..	..	..	..	2
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	1	..	..	..	..	..	..	..	1
Infective Hepatitis	75(20)	11(6)	1(1)	6(4)	22(5)	1	1	..	117
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	3	..	3
Leptospirosis	..	..	4	..	..	..	..	..	4
Malaria	..	..	1	..	..	..	..	..	1
Meningococcal Infection	..	1(1)	..	..	1	..	..	..	2
Ophthalmia	..	..	..	..	1	..	..	..	1
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	..	..	..	..	..	..	..	..
Puerperal Fever	1(1)	..	..	..	..	..	..	..	1
Rubella	..	10(7)	..	..	12(7)	..	..	..	22
Salmonella Infection	..	..	..	4(3)	5(4)	..	1	..	10
Scarlet Fever	17(7)	25(16)	2(1)	1(1)	8(6)	3(1)	..	..	56
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	2(1)	..	..	..	..	2
Trachoma	..	..	..	..	18(9)	..	1	..	19
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	37(17)	10(8)	7(3)	5(4)	2(2)	4(2)	..	..	65
Typhoid Fever	..	..	1(1)	..	1(1)	..	..	..	2
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from Australian Capital Territory.

Thursday, May 15, at 8.15 p.m., "Chronic Pyelonephritis", lecture to members of the British Medical Association at the British Medical Association's Hall.

Tuesday, May 20, at 8.15 p.m., "Renal Osteodystrophy", lecture to Fellows and Members of The Royal Australasian College of Physicians at the Royal Australasian College of Surgeons.

In Brisbane, Professor Rosenheim will be in residence at the Brisbane General Hospital for the period of his stay. He will visit the Mater Misericordiæ and South Brisbane Hospitals on Friday, May 30. The following lectures will be given in Brisbane:

Wednesday, May 28, at 8.15 p.m., "Renal Failure", at the Edwin Tooth Theatre in the grounds of the Brisbane General Hospital.

Friday, May 30, at 8.15 p.m., "Sensitivity Reaction to Drugs", in the Edwin Tooth Theatre in the grounds of the Brisbane General Hospital.

From June 4 to 7 Professor Rosenheim will attend and participate in discussions at the annual meeting of The Royal Australasian College of Physicians to be held in Sydney during that week. He will depart for England on Saturday, June 7.

Notice.

BRITISH MEDICAL ASSOCIATION, VICTORIAN BRANCH.

Section of Industrial Medicine.

A MEETING of the Section of Industrial Medicine of the Victorian Branch of the B.M.A. will be held on Tuesday, May 20, at 8.15 p.m., and will take the form of a plant visit to "Repco Universal", at the corner of Centre Road and Nepean Highway, East Brighton. There is an oil acne dermatitis hazard in this industry and Dr. Ian Stahle will discuss the pathology and treatment of this condition. A demonstration will be given by the Industrial Hygiene Division on a subject of industrial medical interest. All members of the Association are welcome. Supper will be served.

N.S.W. SPORTS MEDICINE ASSOCIATION.

THE next meeting of the New South Wales Sports Medicine Association will be held in the Council Room of the National Fitness Building, corner of Bent and Macquarie Streets, Sydney. This room is adjacent to the National Fitness Council Library, on the second floor. It will take place on Wednesday, May 21, at 8 p.m.

Dr. Alan Barry will lecture on "Psychology in Sports Performance".

All members of the medical profession are cordially invited, and any inquiries concerning this meeting or the Association, may be addressed to the Secretary, Dr. B. Towers, 211 Willarong Road, Caringbah.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Ham, John Mackenzie, M.B., B.S., 1957 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown, New South Wales.

Fisher, Harold Anthony, M.B., B.S., 1956 (Univ. Sydney), 53 Anderson Avenue, Panania, New South Wales.

Leitch, Austen Harding, M.B., B.Ch., 1950 (Univ. Witwatersrand), Base Hospital, Tamworth, New South Wales.

Raneri, Pietro, M.D., 1948 (Univ. Messina), registered in accordance with the provisions of Section 17 (2B) of the *Medical Practitioners Act*, 1938-1957, 198 Flood Street, Leichhardt, New South Wales.

Lacey, Richard Grove, M.B., B.S., 1954 (Univ. London), D.A., R.C.P. & S., 1958, 55 Hanbury Street, Mayfield, Newcastle, New South Wales.

Deaths.

THE following death has been announced:

TAIT.—Leslie Gordon Tait, on April 27, 1958, at Sydney.

Diary for the Month.

MAY 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAY 16.—New South Wales Branch, B.M.A.: Ethics Committee.
MAY 20.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 21.—Western Australian Branch, B.M.A.: General Meeting.
MAY 22.—Victorian Branch, B.M.A.: Executive Meeting.
MAY 22.—New South Wales Branch, B.M.A.: Clinical Meeting.
MAY 23.—Queensland Branch, B.M.A.: Council Meeting.
MAY 27.—New South Wales Branch, B.M.A.: Hospitals Committee.
MAY 28.—Victorian Branch, B.M.A.: Council Meeting.
MAY 29.—South Australian Branch, B.M.A.: Clinical Meeting.
MAY 29.—New South Wales Branch, B.M.A.: Branch Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales. Anti-Tuberculosis Association of New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this Journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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